DRAFT FINAL REPORT

SANTA CLARA CRITERIA AIR POLLUTANT BENEFIT ANALYSIS

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1.0 INTRODUCTION

1.1 OVERVIEW OF THE PROJECT PURPOSE AND APPROACH

The U.S. Environmental Protection Agency, Office of Policy Analysis, Regulatory Integration Division, is presently working with California state and local officials to prepare a risk assessment/risk management analysis for Santa Clara County, California. This report presents estimates of current damages associated with ambient levels of criteria air pollutants in Santa Clara County.

In a previous research effort conducted for the California Air Resources Board (CARB), Rowe et al. (1986) examined the health and welfare effects of selected criteria air pollutants in four major California air basins. Rowe et al. estimated the benefits of California's air quality programs by using the best available information to calculate health and welfare effects that have been avoided since the inception of the CARB programs. Rowe et al. measured health effects in terms of the number of cases of morbidity and premature mortality, with illustrative dollar values also given for each type of health effect. Rowe et al. measured welfare impacts to visibility, materials, agriculture, and forests in dollars.

The analysis reported here uses a similar approach to estimate selected health and welfare impacts for selected criteria pollutants in Santa Clara County. Damage functions that relate health and welfare impacts to ambient levels of pollutants are taken from the available literature. In some cases the damage functions used in the previous analysis are updated with new information. Sufficient information is provided in this report for the reader to understand the sources of the selected damage functions, but for a more complete review of the literature, the reader is referred to the CARB report and other references provided.

Air quality conditions in Santa Clara County indicate that the particulate matter (TSP and PM_{10}), ozone and carbon monoxide concentrations currently exceed the California or Federal standards at one or more monitors. Health effects, increased rates of mortality and morbidity, are estimated for each of these pollutants. The existing sulfur oxide and nitrogen oxide concentrations are in

compliance with current required air quality control levels, and are therefore excluded from the analysis because impacts of levels below the standards are likely to be relatively small. Ambient lead levels in the county are also below the standards, but the effects of gasoline lead emitted from motor vehicles are included in the analysis because multiple sources of lead mean that health effects could be occurring even if ambient levels are low.

Different approaches for estimating the health effects of lead and carbon monoxide are used in this analysis than were used by Rowe et al. This analysis makes use of data on carboxyhemoglobin and blood lead levels available from the Second National Health and Nutrition Examination Survey (NHANES II), in which Santa Clara County was included. Quantitative estimates of the health effects associated with lead in gasoline are presented, but quantitative estimates of the health benefits of meeting the carbon monoxide standards are not obtained due to limitations of available information.

Welfare effects are estimated for visibility (related to changes in particulate matter) and for materials (related to changes ozone and particulate matter). Rowe et al. found that estimated effects for agriculture and forests were relatively small compared to other health and welfare effects for the four California air basins (including the San Joaquin Valley). For this reason, and because of the limited quantitative information available regarding agriculture and forest effects, these effects were not included in the analysis. This does not mean that there are no such effects occurring in Santa Clara County.

Three alternative comparisons are used to estimate damages for each of the pollutants included in the analysis: (1) the damages of current levels as compared to the Federal primary ambient air quality standard, (2) the damages of current levels as compared to California standards, and (3) the damages of current levels as compared to the minimum levels that have been measured in the county in recent years. The first two alternatives presume there is no significant damage below the current standards. The third alternative is considered because of continued scientific controversy regarding the existence of some health and welfare risks below current standards, which is supported in some recent scientific work. Due to the limited evidence concerning effects below the standards, the estimates for the third alternative are more uncertain

than for the other two alternatives, and should be regarded as upper bound estimates. Health estimates for the third alternative can be interpreted as estimates of the health benefits of preventing all manmade emissions of these pollutants, because significant health effects are not expected below these pollution levels.

1.2 GENERAL CAVEATS

The estimates presented in this report are based on the best available evidence (in the judgment of the authors) concerning the effects of air pollutants on the health and welfare of the public. A great deal of uncertainty and controversy remain about much of the research on which these estimates are based. To address this uncertainty, upper and lower range estimates are presented when there is sufficient information in the literature to suggest a likely range. These are not intended as absolute upper and lower bounds, but as ranges within which the actual damages are likely to fall. When no upper and lower ranges are presented for a particular health effect, this indicates more uncertainty rather than less.

The categories of damages presented in this report are not all-inclusive, because quantitative evidence is not available for every effect suspected of being associated with these pollutants. For example, there is some evidence that increased incidences of chronic respiratory illnesses may be associated with exposures to elevated levels of particulate matter and/or ozone, but there is considerable doubt about whether such an association has been adequately demonstrated and insufficient quantitative evidence is available to estimate the potential magnitude of such effects. These types of omissions suggest that the results of the analysis are likely to understate, rather than overstate, the estimated damages of current pollution levels.

Either 1984 or 1985 was used as the baseline level for each of the pollutants. The year in which levels of that pollutant were highest was selected in each case. Thus, 1985 was selected as the baseline year for particulate matter and 1984 as the baseline year for ozone. The estimated health and welfare effects are therefore somewhat higher than would be expected to occur each year on average.

1.3 POPULATION ESTIMATES FOR SAN JOSE AND SANTA CLARA COUNTY

The population base year used for the analysis is 1985. Results are presented for the total county and allocated between San Jose and the remainder of the county. The 1985 population for the county was estimated at 1,398,500 by the U.S. Census. For the city of San Jose, the most recent U.S. Census estimate available was 686,178 for 1984. This estimate represents an average annual increase of 2.2% over the 1980 estimate of 629,400. The 1984 city estimate was therefore increased by 2.2% and subtracted from the 1985 total county estimate to obtain a 1985 estimate for the remainder of the county. The following 1985 population estimates were obtained.

San Jose	701,274
Remainder of County	697,226
Total County	1,398,500

1.4 POLLUTION LEVELS IN SANTA CLARA COUNTY COMPARED TO OTHER AREAS

Representative 1984 levels of ozone, particulate matter (TSP), and carbon monoxide (CO) are shown for several cities in the Bay Area and in the U.S. in Table 1-1 These are presented to give an idea how pollution levels in Santa Clara County compare to other parts of the Bay Area and the U.S. The highest levels of each of these pollutants in the Bay Area occur in Santa Clara County, although levels almost as high do occur in some cities in other counties. This means that damages due to these pollutants would be expected to be similar, but somewhat lower, in many other parts of the Bay Area. For comparison, levels of these pollutants measured in a few major U.S. cities are also shown in Table 1-1 These are similar in many cases to the levels that occur in Santa Clara County, although higher levels of each of the pollutants occur in one or more of the selected cities.

Table 1-1

1984 Criteria Pollutant Levels for

Selected Cities

	1984 Ozone (highest hour-ppm)	1984 TSP (annual geometric mean-µg/m^3)	1984 CO (highest 8-hour average-ppm)
Selected Bay Area Cities			
San Jose[a]	.16	79	12
Los Gatos[b]	.17	--	--
Fremont	.15	50	5
Livermore[c]	.15	56	4
Richmond	.09	56	5
Pittsburg	.16	57	5
Vallejo[d]	.14	41	10
Napa	.11	50	7
Santa Rosa	.09	37	5
San Rafael	.11	56	6
San Francisco[e]	.10	60	8
Redwood City	.11	44	6
Selected U.S. Cities			
Los Angeles	.29	105 (Lynwood)	19 (Lynwood)
New York City	.17	64	15
Washington D.C.	.14	64	14
Chicago	.15	85	11
Denver	.12	142	20

a Highest annual average TSP and 8-hour CO in the Bay Area.
b Greatest number of ozone exceedences in Bay Area.
c Greatest number of ozone exceedences in Bay Area outside Santa Clara County.
d Highest 8-hour CO in Bay Area outside Santa Clara County
e Highest annual average TSP in Bay Area outside Santa Clara County

Sources: California Air Resources Board, "Summary of 1984 Air Quality Data," Sacramento, California.
U.S. Environmental Protection Agency, "National Air Quality and Emissions Trends Report 1984," Research Triangle Park, North Carolina.

1.5 PRIMARY SOURCES OF AIR POLLUTION EMISSIONS

Table 1-2 shows the primary sources of emissions of particulates, reactive organics, nitrogen oxides, and carbon monoxide, based on a 1982 emissions inventory for the Bay Area. Many other sources also exist, but those listed in Table 1-2 represent 85-95% of all emissions of these pollutants. Reactive organics and nitrogen oxides lead to the formation of ozone in the presence of sunlight. The single largest source of ozone precursors and carbon monoxide is motor vehicles, which contribute a slightly larger share in Santa Clara County than in the Bay Area as a whole. Significant sources of ozone precursors also include combustion of fuels in homes, industries, and businesses, and evaporation of solvents during commercial and domestic use. Petroleum refining is a significant source of ozone precursors in other parts of the Bay Area, but not in Santa Clara County. The largest sources of ambient particulates are paved and unpaved roads, construction activity, and miscellaneous industry activity. Motor vehicles and fuel combustion are also important sources. It should be noted that roads and construction activity, although the predominant sources of particulate as measured by tons emitted, tend to produce relatively larger size particles. It is, however, the smaller particles that are believed to have the most significant impact on human health. Motor vehicles and fuel combustion would be relatively larger sources if only small particles were considered.

Table 1-3 shows the percentage of total emissions in the Bay Area that are attributed to sources in Santa Clara County. With the exception of sulfur dioxide, these are roughly proportional to the population of Santa Clara County relative to the Bay Area as a whole. This is primarily due to the absence of petroleum refining activities in Santa Clara County.

1.6 REFERENCES

Rowe, R.D., L.G. Chestnut, D.P. Peterson, C. Miller, R.M. Adams, W.R. Oliver and H. Hogo. 1986. The Benefits of Air Pollution Control in California. Report to the California Air Resources Board, by Energy and Resource Consultants, Inc., Boulder, CO.

Table 1-2

Primary Sources of Air Pollution Emissions

	Percentage of Total Emissions in Tons							
	Particulates		Reactive Organics		Nitrogen Oxides		Carbon Monoxide	
	Bay Area	Santa Clara	Bay Area	Santa Clara	Bay Area	Santa Clara	Bay Area	Santa Clara
Construction & Demolition	19%	24%						
Miscellaneous Industry	20%	14%	3%	5%	1%	0%		
Paved & Unpaved Roads	39%	43%						
Motor Vehicles	6%	7%	43%	47%	56%	75%	83%.	85%
Combustion of Fuels	5%	5%	2%	2%	21%	15%		
Petroleum Refining	1%	0%	8%	0%	10%	0%		
Solvent Usage			28%	30%				

Based on the 1982 Emissions Inventory by the Bay Area Air Quality Management District.

Table 1-3
Percentage of Total Bay Area Emissions (in Tons)
in Santa Clara County

	Percent of Total Bay Area
Population	25%
Particulates	24%
Reactive Organics	24%
Nitrogen Oxides	20%
Carbon Monoxide	26%
Sulfur Dioxide	8%

Based on the 1982 Emissions Inventory by the Bay Area Air Quality Management District

2.0 HEALTH EFFECTS ASSOCIATED WITH PARTICULATE MATTER

2.1 INTRODUCTION

Health effects associated with particulate matter (PM) are estimated for three types of health endpoints: (1) mortality, (2) emergency room visits, (3) and days of acute condition (restricted activity days). Information on estimated quantitative relationships between these health effects and exposures to particulate matter are based on epidemiological studies that have found a significant statistical relationship. These studies demonstrate an association but do not prove causation, and the underlying biological mechanisms that might be occurring are not well understood. These issues are discussed at length in the EPA criteria document for particulate matter (U.S. EPA, 1982), but, need to be noted here because they add uncertainty to the estimates presented. This is addressed to some extent by showing upper and lower ranges of the estimates. Health effects associated with PM are presented in terms of the number of cases expected and in terms of dollar estimates of the economic impact of these health effects.

The most commonly used measure of particulate matter in the air in the U.S. has been total suspended particulates (TSP). This measure, however, includes particles that are too large to be inhaled deeply into human lungs. Therefore, measures of the smaller components of TSP are beginning to be used. California now has a standard defined in terms of PM_{10}, which is a measure of all particles 10 microns in diameter or smaller. The U.S. EPA has proposed a change to a PM_{10} standard rather than a TSP standard. In this analysis we must consider both measures because most of the health effects research that has been conducted has used the TSP measure due to availability of data.

The following sections describe the quantitative evidence on the health effects of PM selected for use in this analysis, the dollar estimates used, the alternative levels of PM considered, the approach used to estimate population exposures to PM for the Santa Clara County population, and the results of the analysis. The health effect is calculated by multiplying the change in air

quality, the health risks associated with that change in air quality, and the exposed population.

2.2 QUANTITATIVE EVIDENCE OF PM HEALTH EFFECTS

Mortality

There are two types of epidemiological studies that have found an association between ambient PM levels and human mortality rates. One type of study (time-series) has looked at changes in mortality rates from day to day in an area as PM levels fluctuate, and the other type of study (cross-sectional) has looked at differences in mortality rates across cities with different PM levels. In both cases a significant association between PM and mortality rates has been found by several different authors. Questions continue to be raised about whether these studies have adequately controlled for potential confounding factors and whether the results can be interpreted as evidence of a causal relationship between PM and mortality (U.S. EPA, 1982). For this reason, we have selected zero as a plausible lower bound estimate of the number of deaths associated with exposures to PM.

Lave and Seskin (1977) conducted an analysis of the relationship between PM and mortality rates for 100 U.S. SMSAs in 1960 and 1969. They found a significant positive'relationship for both TSP and sulfates, but the work has been criticized because control variables such as smoking and diet were not included. Chappie and Lave (1982) used 1974 data for 104 U.S. SMSAs adding diet, smoking and occupational variables to those used by Lave and Seskin, and found significant pollution effects of a comparable magnitude.

Evans et al. (1984) conducted a re-analysis with the original Lave and Seskin data, adding more potential confounding variables and correcting for some coding errors in the original data. Their results indicate a significant relationship between mortality rates and TSP and/or sulfates that is about half the magnitude of the relationship estimated by Lave and Seskin. These results are used in this analysis as the upper range estimate. We selected the TSP coefficient estimated when sulfates are excluded from the equation for this analysis, since

we are focusing on changes in TSP. This coefficient will probably reflect the effect of both TSP and sulfates on mortality and is appropriate for this analysis because when sulfate levels change, TSP would also change. The following equation was used, based on Evans et al. results:

Change in annual deaths/100,000 = .338 * change in TSP (2-1)

where:

TSP = annual arithmetric mean in $\mu g/m^3$.

The best estimate of deaths associated with PM is based on analysis of data from London for 14 winters, 1958/59 through 1971/72. A significant relationship was found between daily deaths and daily levels of British Smoke (BS), a measure of PM that is somewhat different than TSP. These data have been analyzed by Mazumdar et al. (1982), Ostro (1984, 1985), and Schwartz (1986). In its simplified form, the estimated relationship is as follows:

Change in daily deaths in London = .0881 * change in daily BS. (2-2)

To convert this to an equation comparable to Equation (2-1), several adjustments were made. The California Air Resources Board staff paper on PM (CARB, 1982) reports that BS is approximately 55% of TSP. The approximate population in London during this period was 8 million. Equation (2-2) must also be multiplied by 365 to convert it to an annual estimate of deaths. Thus, the London data suggest the following estimates of annual deaths:

Change in annual deaths/100,000 = .0881 * (100,000/8,000,000)
* .55 * 365 * change in TSP

= .221 * change in TSP (2-3)

where :

TSP = annual arithmetric mean in $\mu g/m^3$.

These mortality results from Evan et al. and others are presented in terms of annual deaths per 100,000 people attributable to PM exposures, but it should be noted that everyone in the exposed population could not be expected to face the same risk of premature death. The earlier work by Lave and Seskin indicated that the majority of the deaths would be expected to occur for individuals aged 65 and over. Those with chronic illnesses would also be expected to be at greater risk than average.

Emergency Room Visits (ERV)

Samet et al. (1981) analyzed the relationship between emergency room visits (ERV) and TSP levels in Steubenville, Ohio. Daily ERV at the larger and more urban of the two hospitals in the area were matched with daily levels of TSP, SO_2, and NO_2 for March, April, October, and November of 1974-1977. Visits were regressed on maximum temperature and each of the pollutants in separate estimations. The TSP and SO_2 coefficients were statistically significant in separate equations, but the high correlation between these two pollutants (.69) means that their effects cannot be separated. To the extent that SO_2 may be affecting ERV, applying the Samet et al. findings to Santa Clara County may overstate expected ERV because the SO_2 levels in Santa Clara County are lower than in Steubenville. There is, however, no strong evidence in the literature to suggest that SO_2 has an independent effect on ERV.

The estimated coefficient for daily TSP in a linear regression of daily ERV on TSP and temperature was .011. The authors do not report a standard error but report that the coefficient was significant at the 95% level, but not at the 99% level. This means that the t-statistic is between 1.960 and 2.576. Since the coefficient divided by the standard error equals the t-statistic, the standard error must be between .0043 and .0056. The 95% confidence interval for the estimated TSP coefficient ($\pm$ 1.960 * the standard error) is therefore between $\pm$ .008 and $\pm$ .011. For this analysis, $\pm$ .010 was selected for calculating the upper and lower ranges.

The coefficient must be divided by the Steubenville population to obtain an estimate of per capita ERV that can be applied to Santa Clara County. The authors report a population of 31,000 at the time of the study. Because the

estimated relationship between ERV and TSP is linear, the annual change in ERV is 365 times the daily change. The following calculations were therefore made to estimate the change in annual ERV:

Lower change in annual per capita ERV = (.001/31,000) * 365 * change in TSP = .000012 * change in TSP (2-4)

Best change in annual per capita ERV = (.011/31,000) * 365 * change in TSP = .00013 * change in TSP (2-5)

Upper change in annual per capita ERV = (.021/31,000) * 365 * change in TSP = .00025 * change in TSP (2-6)

where:

TSP = annual arithmetic mean in $\mu g/m^3$.

Because the Samet et al. study was conducted in an area where the components of TSP may be significantly different than in Santa Clara County (there is considerable steel industry activity in Steubenville), the error involved in applying these results to changes in TSP in Santa Clara County is potentially greater than may be indicated by the upper and lower ranges.

Restricted Activity Days

Estimates of an association between restricted activity days (RAD) and PM were based on Ostro (1987). RAD include days spent in bed, days missed from work, as well as days when activities are restricted to a minor degree due to a health condition. Ostro estimated a relationship between RAD in a two-week period and fine particles (FP, diameter less than 2.5 microns) in the same two-week period for 49 metropolitan areas in the U.S. Separate regression estimates were obtained for 6 years, 1976-1981. The RAD data were from the Health Interview Survey conducted annually by the National Center for Health Statistics. The FP data were estimated from visual range data available for airports in each area. Fine particles have a more significant impact on visual range than do larger suspended particles, so there is a direct relationship between visual range and

the amount of FP in the air. A statistically significant result was found in each year. This study follows and supports the findings of an earlier study (Ostro, 1983) where a significant relationship was found between RAD and TSP measured at stationary monitors in each area.

The form of the estimated relationship was such that the coefficient for FP gives the percentage change in RAD associated with a unit change in FP. For any two-week period i, the change in RAD would be:

$$\text{Change in RADi} = b * \text{RADi} * \text{change in FPi} \qquad (2\text{-}7)$$

where :

b = estimated coefficient.

Equation (2-7) can be converted to give an annual estimate of changes in RAD if we accept the following assumptions.

(1) RADi = RADa/26, where RADa is the annual average number of RAD. This assumption is necessitated by data availability since RAD data for every two-week period of the year are not available.

(2) $\left(\sum_{i=1}^{26} \Delta \text{FPi}\right)/26 = \Delta$ FPa, where Δ FPa is the change in the annual average FP. This assumption is valid if the number of observations in each two-week period is equal.

Thus, the annual change in RAD can be estimated as follows:

$$\text{Change in RADa} = b * \text{RADa} * \text{change in FPa.} \qquad (2\text{-}8)$$

It is necessary to convert changes in FP to changes in TSP, because changes in PM for this analysis are being defined in terms of changes in TSP (see Section 2.4). Data from the U.S. EPA Inhalable Particle Monitoring Network indicate that for several stations in California, an average ratio of FP to TSP is .24.

Data from the National Center for Health Statistics indicate that the average annual number of RAD per person in the U.S. is about 19 days. Equation (2-8) can thus be written:

Change in RADa/person = b * 19 * .24 * change in TSP (2-9)

where :

TSP = annual arithmetic mean in $\mu g/m^3$.

The estimated coefficient (b) for FP ranged from .003 to .009 over the 6 years for which it was estimated. The estimated coefficients fell into two groups: .003 to .004 and .006 to .009. The upper range estimate for this analysis was selected as the mean of the higher group and the lower range estimate as the mean of the lower group. The best estimate was selected as the mean of all 6 estimates.

Selected Coefficients

Upper	.0076
Best	.0048
Lower	.0034

The estimated number of ERV were subtracted from the estimates of RAD since these would also be considered restricted activity days.

2.3 DOLLAR VALUE ESTIMATES FOR PM MORBIDITY EFFECTS

Dollar estimates of the value of preventing each of the nonfatal health effects were developed, based on available information, to allow comparisons to the costs of pollution control, and to allow some comparison of the relative significance of the different categories of effects, such as health, visibility and materials damage. A modified cost-of-illness approach is used. These estimates probably do not reflect the full value of preventing illness because traditional cost-of-illness estimates do not fully reflect the discomfort and

inconvenience experienced by the individual, but some efforts are made to compensate for this.

Dollar value estimates for RAD are based on an approximate value of time spent ill, which is presumed to be related to the average wage rate. For days when the individual is ill enough to miss work, the value is placed at the average daily wage for California residents. For 1985, this was about $80 (based on data reported for California by the U.S. Bureau of Labor Statistics). Data from the Health Interview Survey by the National Center for Health Statistics indicated an average of 19 RAD per person per year and an average of 5 work loss days per person per year. If we assume that a proportional number of weekend days are affected to the point where very little activity is possible, similar to a work loss day, then we can say that 7 (5 * 365 total days/260 work days = 7) of the 19 RAD are severe enough to keep a person home from work or significantly restrict recreational activity. If this degree of restriction is valued at the daily wage, and days restricted to a more minor degree are valued at one-fourth the daily wage, then the average value for a RAD is $42. This value should be thought of as a plausible figure, rather than a concrete estimate.

Dollar value estimates for ERV are based on the value of time lost (presumably one day) and average costs of a visit to an emergency room. The value of time was again placed at the average daily wage of $80. Average medical expenditures (including the amount covered by insurance) per ERV is based on data reported by the California Health Facilities Commission. For fiscal year 1979-1980, total reviews for out-patient services were $1,518,267,051 and total out-patient visits were 11,642,715, implying an average cost of about $130 per out-patient visit. Inflated to 1985 dollars this is about $180. Thus, the dollar value estimate for an ERV is $260.

2.4 CHANGES IN TSP FOR THE DIFFERENT SCENARIOS

The health effects associated with PM (as well as materials damage and visibility discussed in subsequent chapters) were calculated for changes in PM from levels that currently occur in Santa Clara County. For this purpose, a

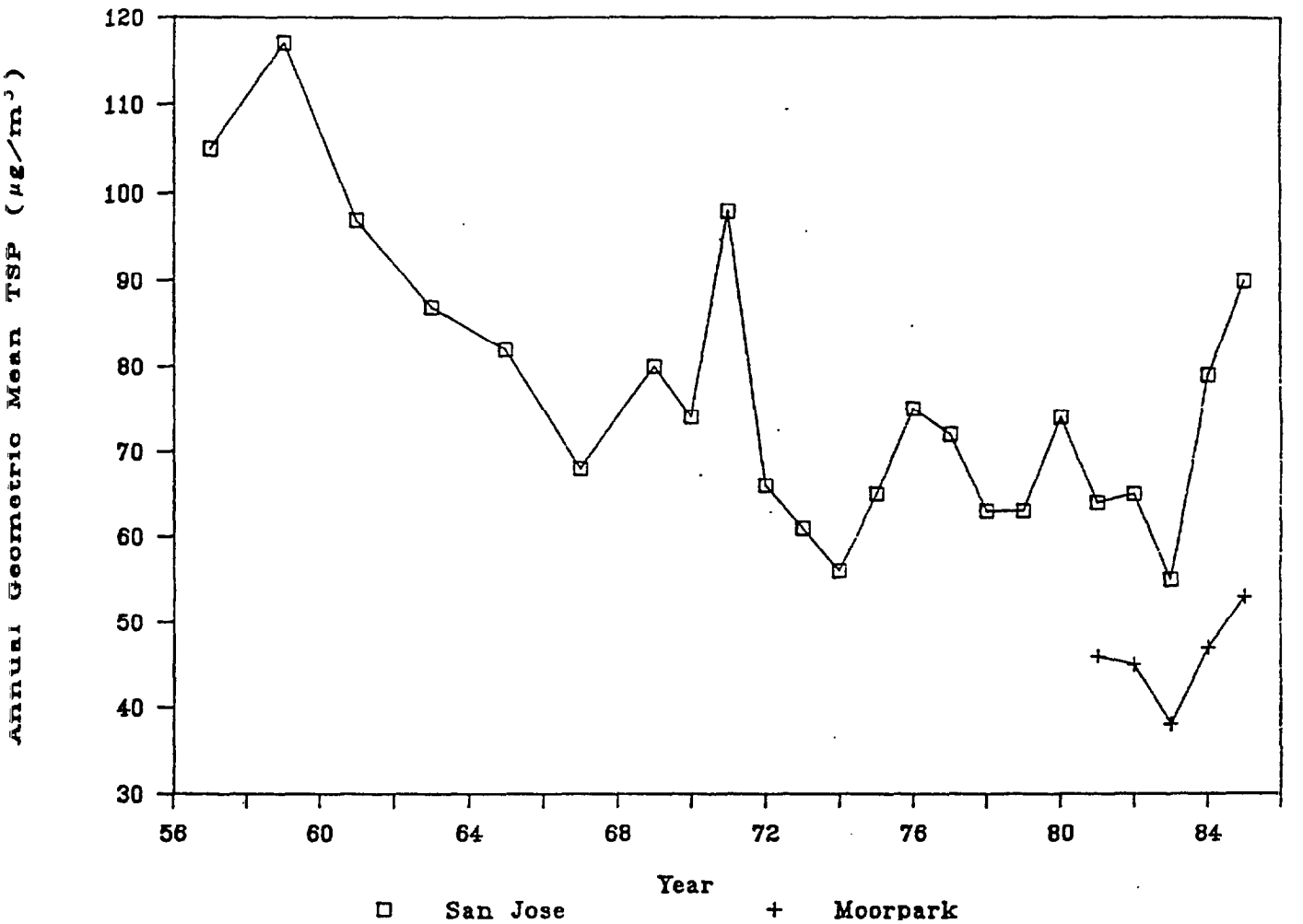
baseline of actual PM levels was defined in terms of the TSP measures needed in the health calculations. Alternative levels of PM were defined by estimating what these measures of TSP would be if (1) the Federal TSP standard were met, (2) the California PM10 standard were met, or (3) levels of TSP as low as have been measured at any monitor in the county in recent years were attained.

Baseline TSP

1985 levels of TSP were used as the baseline in these calculations. This the most recent year for which complete data were available, and showed somewhat higher levels than 1984. Figure 2-1 shows the annual geometric mean TSP measured in San Jose from 1957 to 1985. There is an overall downward trend with a few relatively high spikes. The level in 1985 is the second highest level measured in the 1970s and 1980s. Levels measured at the Moorpark monitor are shown for available years, 1981-1985. These are consistently lower than those measured at the 4th Street monitor, but they show a similar year-to-year pat tern. Because the 4th Street monitor is located in the downtown area of the city, TSP levels measured at this monitor were used for the census tract groups located close to the 4th Street monitor (which amounted to about 4% of the county population). Moorpark levels were used for the remainder of the county.

The first column of Table 2-1 shows the geometric mean, arithmetic mean, and second high of the 24-hour readings of TSP at the two monitors in 1985. The geometric standard deviation is estimated rather than actual. To estimate changes in TSP under different scenarios it was necessary to estimate how the geometric standard deviation would change as the geometric mean changed. The geometric standard deviation is needed to estimate, for example, what the second high reading in the year would be expected to be given a specific annual geometric mean and vice versa. It is also needed to convert the geometric mean to an arithmetic mean, which is used in the health effects calculations. A regression was estimated relating changes in the measured geometric standard deviation (GEOSD) to the measured geometric mean (GEOMEAN) using data from the two monitors for 1981-1985. The following equation was obtained:

Figure 2-1
TSP Levels in San Jose 1957 - 1985



NOTES:

1. Data are for every other year 1957-1967 and for every year 1969-1985.
2. San Jose monitor location changed in August 1972 from W. Alma St. to 4th St. Level for 1972 is average of the two stations weighted for the number of observations at each station.
3. Measurement technique changed from cellulose filters to glass fiber filters in 1976. Data from Bay Area monitors suggest the glass filter reads 3-4 $\mu g/m^3$ higher.

Table 2-1

Estimated Levels of TSP for Santa Clara County Monitors Under Alternative Scenarios

	1985 Baseline		Levels That Would Meet Federal TSP Standards[c]	Change From Actual 1985 to Meet Federal Standard	Levels That Would Meet California PM10 Standards[d]	Change from Actual 1985 to Meet California Standards	Observed Low TSP Levels[e]	Change from Actual 1985 to Observed Low Levels
	Actual	Adjusted[b]						
4th Street Monitor								
Annual Geometric Mean	90	75	75	15	46	44	38	52
Annual Arithmetic Mean	102	81	81	21	48	54	39	63
Second High 24-hour	269	220	220	49	95	174	74	195
Annual Geometric Standard deviation[a]	1.6	1.5	1.5	1.3	1.3			
Moorpark Monitor								
Annual Geometric Mean	53	53	75	0	46	7	38	15
Annual Arithmetic Mean	59	59	81	0	48	11	39	20
Second High 24-hour	107	107	220	0	95	12	74	33
Annual Geometric Standard deviation*	1.4	1.4	1.5		1.3		1.3	

a Geometric standard deviation based on estimated Equation 2-8.
b Adjusting 4th St. to (1.7) * Moorpark due to suspected elevated levels in 1984-85.
c Federal standard is 75 $\mu g/m^3$ for the annual geometric mean. This is more constraining than the Federal 24-hour standard, given the historic geometric standard deviation for TSP in Santa Clara County. Arithmetic mean and second high are estimated using the 75 $\mu g/m^3$ annual geometric mean and the estimated geometric standard deviation (see text).
d California standard for 24-hours in 100 $\mu g/m^3$ TSP (converted from PM10) not to be exceeded. This is more constraining than the California standard for the annual geometric mean given the historic geometric standard deviation for TSP in Santa Clara County. The second high and two means are estimated assuming the highest hour is 100 $\mu g/m^3$ (see text).
e Annual geometric mean at Moorpark in 1983 was 39 $\mu g/m^3$.

$$GEOSD = 1.05 + .0061 * GEOMEAN \quad (2\text{-}10)$$
$$(.15)\ (.0025)$$
$$R^2 = .43$$

Standard errors of the estimated coefficients are in parentheses. The coefficients are statistically significant at 95% confidence. Table 2-2 shows the data on which Equation (2-8) is based and the predicted standard deviations implied by the estimated coefficients.

Staff at the San Francisco Air Quality Management District who operate the monitors in Santa Clara County indicated that levels at the 4th Street station have been elevated in 1984 and 1985 due to construction in the vicinity of the monitor. The data for 1981-1983 indicate that the geometric means at the Moorpark monitor were about 70% of the means at the 4th Street monitor. For 1984 and 1985 this fell to about 60%. For comparison purposes, an adjusted baseline for the 4th Street monitor was calculated by multiplying the geometric mean at the Moorpark monitor by 1/0.7. This and the other TSP measures that would be expected with an annual geometric mean of (1/0.7) * 52.8 are shown in the second column of Table 2-1. Only the 4th Street monitor is changed.

Meeting the Federal TSP Standard

The third column in Table 2-1 shows the estimated levels of TSP that would meet the Federal standard. The Federal primary TSP standard is a geometric mean of 75 $\mu g/m^3$, which is not to be exceeded, and a 24-hour level of 260, which is not to be exceeded more than once a year. When the annual geometric mean is 75 $\mu g/m^3$, the predicted geometric standard deviation from Equation (2-10) is 1.5. This means a value at the 99.45 percentile of the lognormal distribution of 220 $\mu g/m^3$. The 99.45 percentile can be considered an estimate of the second high value for the year (363 days/365 days = 99.45%). When the annual geometric mean is 75, the second high day would thus be below the 24-hour standard (second high not to exceed 260 $\mu g/m^3$) based on this estimation procedure. Therefore, the Federal annual average standard is constraining, given the historic distribution of TSP levels in Santa Clara County. The arithmetic mean and the second high 24-hour level given in column 3 in Table 2-1 are estimates of these measures

Table 2-2

Estimated Relationship Between Geometric Mean and Geometric Standard Deviation

Monitor	Year	Annua1 Geometric Mean	Actual Geometric Standard Deviation	Predicted Geometric Standard Deviation (from Equation 2-10)
Moorpark	1983	38.4	1.3	1.28
Moorpark	1982	45.1	1.4	1.29
Moorpark	1981	45.9	1.2	1.33
Moorpark	1984	47.1	1.4	1.34
Moorpark	1985	52.8	1.6	1.37
4th street	1983	54.8	1.3	1.38
4th street	1981	64.3	1.3	1.44
4th Street	1982	65.3	1.4	1.45
4th Street	1984	78.7	1.5	1.53
4th Street	1985	90.2	1.7	1.60

that would be expected when the annual geometric mean equals 75 $\mu g/m^3$. The arithmetic mean associated with the geometric mean of 75 is calculated using the following formula (from EPA 1971):

$$\ln m = \ln mg + .5 * (\ln sg)^2 \qquad (2\text{-}11)$$

where :

m = arithmetic mean

mg = geometric mean

sg = geometric standard deviation

As shown in Table 2-1, TSP levels at the Moorpark monitor are below the Federal primary standard. Estimates of the change required to meet the Federal standard are therefore shown as zero in column 4 for the Moorpark monitor. The actual 1985 levels at the 4th Street monitor exceed the Federal standard, but the adjusted levels just meet the standard.

Meeting the California PM10 Standard

The estimated TSP levels that would be associated with attainment of the California PM_{10} standards are shown in the fifth column of Table 2-1. The California standard for particulate matter is written in terms of particles less then 10 microns (PM_{10}) rather than total suspended particles. The annual geometric mean, not to be exceeded, is 30 $\mu g/m^3$ and the 24-hour level, not to be exceeded, is 50 $\mu g/m^3$.

Currently available damage functions for particulate matter are based on TSP rather than PM_{10} data. To estimate the benefits that would occur if the California PM_{10} standard were met it was necessary to estimate what TSP levels would be if the PM_{10} standards were met. It was presumed that PM_{10} would remain a constant percentage of TSP. An estimate that PM_{10} is roughly 50% of TSP was used for this purpose. This value was obtained from the EPA staff paper on PM_{10} (EPA, 1986), and was confirmed with 1985 PM_{10} and TSP data available for the 4th Street monitor in San Jose (which showed an average ratio of .51).

Using the .5 ratio, the TSP levels that would be equivalent to the California PM_{10} standards are:

Annual Geometric Mean 60 $\mu g/m^3$

Highest 24-Hour Not to Exceed 100 $\mu g/m^3$

The 99.7 percentile of the lognormal distribution was used to estimate the highest 24-hour reading in a year (364/365 = 99.7%). A 99.7 percentile of 100 $\mu g/m^3$ is consistent with a geometric mean of 46 $\mu g/m^3$ and a second high 24-hour level of 95 $\mu g/m^3$, when the geometric standard deviation is 1.33 (which is what would be predicted by Equation 2-10). This means that given the historic distribution of TSP in Santa Clara County, the California 24-hour standard is more constraining than the standard for the annual geometric mean. The TSP levels shown in column 5 of Table 2-1 are the levels predicted to occur when the highest 24-hour TSP level is 100 $\mu g/m^3$.

Low Observed Level of PM

The lowest annual geometric mean TSP during 1981-1985 at either of the monitors in the county was 38 $\mu g/m^3$ at Moorpark in 1983. This level is also typical of the low end of the TSP data used in studies estimating relationships between TSP and health outcomes. For example, 39 (arithmetic mean) is two standard deviations below the mean in the Evans et al. data used to examine the relationship between annual mean TSP and mortality rates in several U.S. cities. It would therefore be inappropriate to predict changes in health effects at levels of TSP much below this. Based on Equations 2-10 and 2-11, the arithmetic mean is 39 and the second high is 74 when the geometric mean is 38. These are shown in the seventh column in Table 2-1.

Proposed Federal PM10 Standard

The U.S. EPA is currently considering adoption of a PM_{10} standard. The levels for this standard have not been finalized, but there is a possibility that the San Jose monitor might not be in compliance with the new standard, given the levels that are being considered for the standard. PM_{10} levels measured at the San Jose 4th Street monitor in 1985 are (based on 61 observations):

Annual Arithmetic Mean	52.5
First Highest 24-hour	181
Second Highest 24-hour	164

Since the Federal PM_{10} standard is not yet final, no estimates have been made concerning potential benefits of meeting the proposed standards for this analysis.

2.5 RESULTS

The estimated numbers of cases that would be prevented for each of the potential reductions in PM are shown in Tables 2-3 and 2-4. Table 2-3 gives the estimates calculated with the actual 1985 baseline, and Table 2-4 gives the estimates calculated with the adjusted 1985 baseline.

The differences between the estimates in Tables 2-3 and 2-4 are most significant for the Federal standard scenario, because with the adjusted baseline, the county is in compliance with the Federal standard. In general, the difference between the adjusted and actual baselines (based on the best estimates) is about 3 deaths, 175 ERV and 30,000 RAD. Even though the elevated TSP levels at the 4th Street monitor may be temporary, they are indicative of actual levels in the area. The estimates in Table 2-4 should be interpreted not as more accurate than those in Table 2-3, but as estimates of what effects would have been likely had there been no construction activity in the area of the 4th Street monitor.

To give an idea of the significance of the estimates relative to the county population, consider the county total best estimates for meeting the Federal standard. These imply an annual reduction in 1 death for every 466,000 people, 1 ERV for every 8,000 people, and 1 RAD for every 50 people. The county total best estimates for meeting the California standards imply an annual reduction in 1 death for every 35,000 people, 1 ERV for every 600 people, and 2 RAD for every 7 people.

Applying the dollar value estimates discussed in Section 2.3 to the number of nonfatal cases shown in Tables 2-3 and 2-4, gives the dollar estimates for

predicted changes in morbidity associated with potential reductions in PM in Santa Clara County. These are shown in Tables 2-5 and 2-6. The best estimate for meeting the Federal standard (based on the actual baseline) implies about $1 per person as a county-wide average. The best estimate for meeting the California standard is $12 per person as a county-wide average based on the actual baseline, and about $11 per person based on the adjusted baseline. For the California best estimate, about 54% to 58% of the county total is attributed to San Jose.

Table 2-3

Predicted Reductions in Health Effects Associated with Alternative Reductions in Particulate Matter from Actual 1985 Levels

	Number of Cases Reduced		
	Deaths	Emergency Room Visits	Restricted Activity Days (thousands)
San Jose			
Meeting Federal Standard			
Lower	0.	16.	21.3
Best	3.	173.	29.2
Upper	5.	330.	46.4
Meeting California Standards			
Lower	0.	123.	167.0
Best	23.	1353.	228.5
Upper	35.	2583.	363.1
Lowest PM Levels			
Lower	0.	197.	268.0
Best	37.	2171.	366.5
Upper	57.	4144.	582.4
Remainder of County			
Meeting Federal Standards			
Lower	0.	0.	0.2
Best	0.	2.	0.3
Upper	0.	4.	0.5
Meeting California Standards			
Lower	0.	91.	123.1
Best	17.	997.	168.4
Upper	26.	1904.	267.6
Lowest PM Levels			
Lower	0.	165.	223.5
Best	31.	1810.	305.7
Upper	47.	3456.	485.7
Total County			
Meeting Federal Standards			
Lower	0.	16.	21.6
Best	3.	175.	29.5
Upper	5.	334.	46.9
Meeting California Standards			
Lower	0.	214.	290.2
Best	40.	2351.	396.9
Upper	61.	4487.	630.7
Lowest PM Levels			
Lower	0.	362.	491.4
Best	68.	3981.	672.2
Upper	104.	7600.	1068.2

Table 2-4

Predicted Reductions in Health Effects Associated with Alternative Reductions in Particulate Hatter from Adjusted 1985 Levels

	Number of Cases Reduced		
	Deaths	Emergency Room Visits	Restricted Activity Days (thousands)
San Jose			
Meeting Federal Standard			
Lower	0.	0.	0.0
Best	0.	0.	0.0
Upper	0.	0.	0.0
Meeting California Standards			
Lower	0.	107.	145.7
Best	20.	1180.	199.3
Upper	31.	2253.	316.7
Lowest PM Levels			
Lower	0.	182.	246.6
Best	34.	1998.	337.3
Upper	52.	3814.	536.0
Remainder of County			
Meeting Federal Standards			
Lower	0.	0.	0.0
Best	0.	0.	0.0
Upper	0.	0.	0.0
Meeting California Standards			
Lower	0.	90.	122.9
Best	17.	995.	168.1
Upper	26.	1900.	267.1
Lowest PM Levels			
Lower	0.	164.	223.2
Best	31.	1808.	305.3
Upper	47.	3452.	485.2
Total County			
Meeting Federal Standards			
Lower	0.	0.	0.0
Best	0.	0.	0.0
Upper	0.	0.	0.0
Meeting California Standards			
Lower	0.	198.	268.6
Best	37.	2176.	367.4
Upper	57.	4154.	583.8
Lowest PM Levels			
Lower	0.	346.	469.8
Best	65.	3806.	642.7
Upper	99.	7266.	1021.2

Table 2-5
Dollar Values for Predicted Reductions in
Morbidity Associated with Particulate Matter
(Based on Actual 1985 PM Levels)

	Dollar Amount Reduced		
	Emergency Room Visits (thousands)	Restricted Activity Days (thousands)	Total Dollars (thousands)
San Jose			
Meeting Federal Standard			
Lower	4.1	896.6	900.7
Best	45.0	1226.5	1271.4
Upper	85.8	1948.9	2034.7
Meeting California Standards			
Lower	32.0	7015.9	7047.9
Best	351.8	9597.2	9949.0
Upper	671.7	15250.2	15921.9
Lowest PM Levels			
Lower	51.3	11254.2	11305.5
Best	564.4	15394.7	15959.1
Upper	1077.4	24462.5	25540.0
Remainder of County			
Meeting Federal Standards			
Lower	0.0	10.1	10.2
Best	0.5	13.8	14.3
Upper	1.0	22.0	22.9
Meeting California Standards			
Lower	23.6	5171.2	5194.8
Best	259.3	7073.8	7333.1
Upper	495.1	11240.4	11735.5
Lowest PM Levels			
Lower	42.8	9385.3	9428.1
Best	470.6	12838.2	13308.9
Upper	898.5	20400.2	21298.8
Total County			
Meeting Federal Standards			
Lower	4.1	906.7	910.9
Best	45.5	1240.3	1285.8
Upper	86.8	1970.9	2057.7
Meeting California Standards			
Lower	55.6	12187.2	12242.7
Best	611.2	16671.0	17282.1
Upper	1166.7	26490.6	27657.3
Lowest PM Levels			
Lower	94.1	20639.4	20733.5
Best	1035.0	28232.9	29267.9
Upper	1975.9	44862.8	46838.7

Table 2-6
Dollar Values for Predicted Reductions in
Morbidity Associated with Particulate Matter
(Based on Adjusted 1985 PM Levels)

	Emergency Room Visits (thousands)	Restricted Activity Days (thousands)	Total Dollars (thousands)
San Jose			
Meeting Federal Standards			
Lower	0.0	0.0	0.0
Best	0.0	0.0	0.0
Upper	0.0	0.0	0.0
Meeting California Standards			
Lower	27.9	6119.3	6147.2
Best	306.9	8370.7	8677.6
Upper	585.8	13301.3	13887.1
Lowest PM Levels			
Lower	47.2	10357.5	10404.8
Best	519.4	14168.2	14687.6
Upper	991.6	22513.6	23505.2
Remainder of County			
Meeting Federal Standards			
Lower	0.0	0.0	0.0
Best	0.0	0.0	0.0
Upper	0.0	0.0	0.0
Meeting California Standards			
Lower	23.5	5161.1	5184.6
Best	258.8	7059.9	7318.7
Upper	494.1	11218.4	11712.5
Lowest PM Levels			
Lower	42.7	9375.2	9417.9
Best	470.1	12824.4	13294.5
Upper	897.5	20378.3	21275.8
Total County			
Meeting Federal Standards			
Lower	0.0	0.0	0.0
Best	0.0	0.0	0.0
Upper	0.0	0.0	0.0
Meeting California Standards			
Lower	51.4	11280.4	11331.9
Best	565.7	15430.6	15996.3
Upper	1079.9	24519.7	25599.6
Lowest PM Levels			
Lower	90.0	19732.7	19822.7
Best	989.5	26992.6	27982.1
Upper	1889.1	42891.9	44781.0

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3.0 HEALTH EFFECTS ASSOCIATED WITH OZONE

3.1 INTRODUCTION

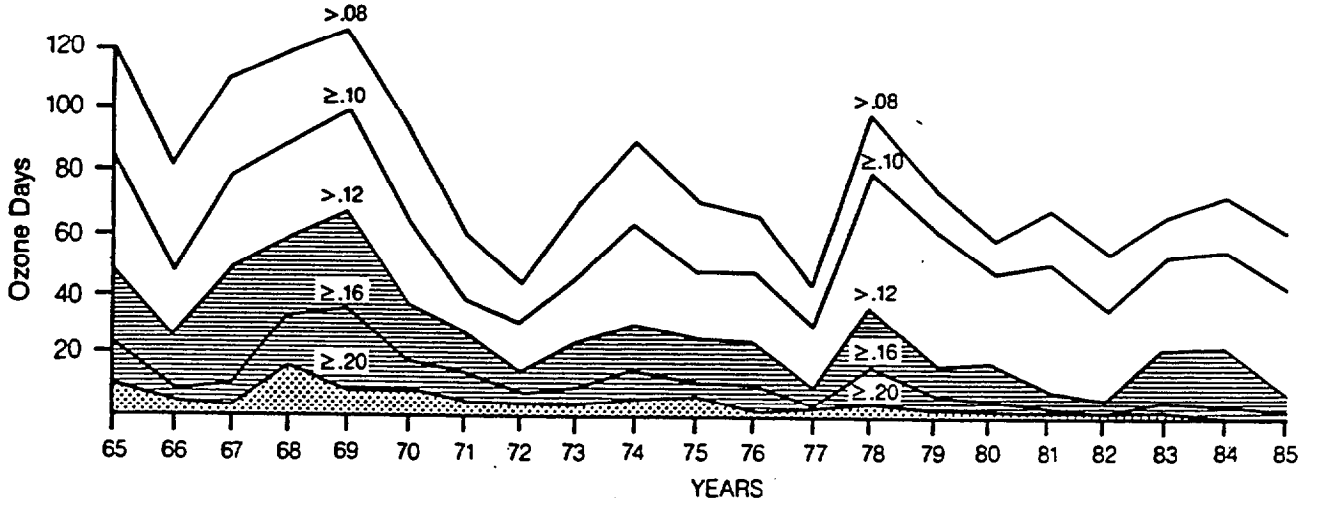
Ozone is a pollutant that forms in the atmosphere in the presence of hydrocarbons, nitrogen oxides, and sunlight. It is the primary component of what is known as photochemical smog. It is believed to be irritating to the human respiratory system. Estimates are presented in this chapter for the effects of ozone on (1) respiratory restricted activity days, (2) asthma attacks, and (3) eye irritation.

Because meteorological conditions play such an important role in the formation of ozone in the atmosphere, ambient ozone levels vary significantly over time. For this analysis we chose 1984 as the baseline year. Figure 3-1 shows the trend in ozone levels in the Bay Area over the period 1965 to 1985. Although the year-to-year fluctuation dominates, there appears to be a downward trend in ozone levels during this period. As Figure 3-1 shows, ozone levels in 1984 were the highest that have occurred in the 1980s. Fairly similar levels also occurred in 1983 but levels in 1982 and 1985 were significantly lower. For example, the number of days exceeding the hourly Federal ozone standard at the San Jose monitor was 9 in 1983, 7 in 1984, and 2 in 1985. The estimates presented in this chapter of reductions in health effects that could be obtained with alternative reductions in ozone are for a year when the ozone levels were higher than sometimes occurs, and therefore probably exceed the average annual reduction in health effects that would be obtained given recent ozone levels.

3.2 QUANTITATIVE EVIDENCE OF OZONE HEALTH EFFECTS

Evidence concerning the health effects associated with ozone is reviewed in detail in the EPA criteria document for ozone (U.S. EPA, 1983) and summarized by Ferris (1978). Clinical studies, in which subjects have been exposed to a known amount of ozone in a controlled setting, have demonstrated fairly conclusively that ozone is irritating to the nose and throat and causes temporary reductions in lung function. These effects have been found in adults with chronic

Figure 3-1
Ozone Levels in the Bay Area 1965-1985



Annual occurrence of days when any station in the Bay Area Air Quality Management District exceeded the indicated ozone levels at an hourly average, in parts per million. The California standard is equal to or greater than .10ppm. The federal standard is greater than .12ppm. The first stage 'Advisory/Alert' level is equal to or greater than .20ppm.

>Greater Than ≥Equal To Or Greater Than

Source: Bay Area Air Quality Management District, Air Currents, Vol. 30, No. 4, April 1987.

respiratory conditions and in healthy children and adults. Quantitative dose-response relationships are difficult to identify from these types of studies, but the findings help support the epidemiological studies that have found a significant relationship between ambient ozone levels and occurrences of respiratory restricted activity days or asthma attacks.

Respiratory Restricted Activity Days

Portney and Mullahy (1986) used the health Interview Survey for 1979 (conducted by the National Center for Health Statistics) to examine the relationship between restricted activity days and ozone levels. To the nation-wide health data, the authors added pollution and weather data for each respondent's residential location. A significant positive relationship was found between restricted activity days due to respiratory illness (RRAD) and ozone levels. The data were for two-week periods. The ozone measure was the two-week average of the daily maximum hour for each location.

The authors tested 5 different specifications of the relationship between ozone and RRAD. In 4 of the 5 specifications, the estimated elasticity of changes in RRAD for adults with respect to ozone ranged from .3 to .6. The mean of all 5 elasticity estimates was .42. This means that a 10% change in ozone is associated with a 4.2% change in RRAD. We selected the estimated coefficients associated with an elasticity of .3 for calculating the lower estimate, and the estimates associated with an elasticity of .5 for the upper estimate. The best estimate is calculated as the midpoint between the upper and lover estimates.

A baseline level of RRAD was needed for the calculations because the change in RRAD predicted by the Portney and Mullahy results is a function of the baseline RRAD as well as the change in ozone. Data from the Health Interview Surveys indicate a national annual average of about 8 RRAD per person. This implies an average of .31 per person every two weeks. The 1980 Census indicates that 72% of the population in Santa Clara County is 18 and over. The RRAD estimates were calculated for adults only because the Portney and Mullahy results are for adults. The estimated percentage of the population with asthma (7.7% according to the California Department of Health Services) was excluded from this calculation since effects of ozone on asthma attacks are considered separately.

Equations 3-1 and 3-2 show the predicted lower and upper changes in RRAD for a change in ozone, based on the Portney and Mullahy results. These are the derivatives of the estimated functions with respect to ozone, evaluated with the .31 estimate of average RRAD in a two-week period.

$$\text{Lower change in RRAD/person} = .31 * 6.883 * \text{change in } 01 \quad (3\text{-}1)$$

$$\text{Upper change in RRAD/person} = .31 * 4.926 * .5 * (01)^{-.5} * \text{change in } 01 \quad (3\text{-}2)$$

where:

01 = the two-week arithmetic average of the daily high hourly ozone reading in ppm

Equations (3-1) and (3-2) were applied to the adult, non-asthmatic portion of the population (about 66%). These equations were evaluated for every two-week period in the 36-week ozone season and then summed over the 18 two-week periods. The lower and upper estimates were thus:

$$\text{Lower change in RRAD} = \sum_{i=1}^{18} (.31 * 6.883 * \text{change in } 01 * .72 * (1-.077) * \text{population})$$

$$= \sum_{i=1}^{18} (1.418 * \text{change in } 01 * \text{population}) \quad (3\text{-}3)$$

$$\text{Upper change in RRAD} = \sum_{i=1}^{18} (.31 * 4.926 * .5 * (01)^{-.5} * \text{change in } 01 * .72 * (1-.077) * \text{population})$$

$$= \sum_{i=1}^{18} (.507 * (01)^{-.5} * \text{change in } 01) \quad (3\text{-}4)$$

Asthma Attacks

Whittemore and Korn (1980) studied individuals (children and adults) with diagnosed asthma who lived in six communities in the Los Angeles area during

1972-1975. Subjects made daily reports of asthma attack occurrence for a 35 week period each year. These were found to be significantly related to daily maximum ozone hour measured at monitors in or close to each community. Their results indicate the following change in the probability of an asthma attack for an increase in ozone, holding other factors constant:

$$\text{Change in Pt} = 1.66 * Pt * (1 - Pt) * \text{change in Od} \qquad (3\text{-}5)$$

where:

Pt = the probability of an asthma attack on day t
Od = daily high ozone hour in ppm.

Schwartz et al. (1985) give a range of estimates of .014 to .028 as the average daily probability of an asthma attack for asthmatics in the U.S., taking into account the different frequencies that occur among the asthmatic population. The midpoint of these two values is substituted for Pt in Equation (3-5). The following equation gives the best estimate of the predicted change in the number of asthma attacks per day for a change in the daily high hourly reading of ozone. An estimate that 7.7% of the population has asthma was obtained for California from the California Department of Health Services. Equation (3-6) was evaluated for each day in the ozone season and then summed over the 36-week period.

$$\text{Best change in asthma} = 1.66 * .021 * (1-.021) * \text{change in } 02 * .077 * \text{population}$$

$$= .00263 * \text{change in } 02 * \text{population} \qquad (3\text{-}6)$$

where:

02 = daily high hourly ozone in ppm.

Upper and lower estimates were based on 95% confidence intervals for the 1.66 coefficient. Whittemore and Korn report that this coefficient is significant at the 99% level. Although they do not report a standard error, the 99%

significance means that the standard error must be .64 or less. The 95% confidence interval is therefore no more than ± 1.25. The 1.66 coefficient ± 1.25 was therefore used to calculate lower and upper estimates.

Lower change in asthma = .41 * .021 * (1-.021) * change in 02 * .077 * population

= .00065 * change in 02 * population

Upper change in asthma = 2.91 * .021 * (1-.021) * change in 02 * .077 * population

= .00461 * change in 02 * population

Eye Irritation

Hammer et al. (1974) found higher rates of self-reported eye irritation in a sample of student nurses in Los Angeles on days with higher ozone levels. Evidence reported by the U.S. EPA (1983) indicates that eye irritation is not caused by ozone, but by another component of photochemical smog that commonly occurs in association with ozone. The estimates of eye irritation presented here as a function of ozone levels therefore rely on the presumption that ozone and the other components of photochemical smog are highly correlated.

Hasselblad and Svendsgaard (1975) reanalyzed the Hammer et al. results. The following summary of their findings is taken from Schwartz et al. (1985). Hasselblad and Svendsgaard estimated a relationship between the probability of experiencing eye irritation on a given day and the daily maximum ozone hour. The probability of an individual experiencing eye irritation at ozone level 0x, measured in parts per hundred million, was estimated as

$$p(0x) = .0407 + (1 - .0407) / (1 + \exp(4.96 - .0907 * 0x)) \quad (3\text{-}7)$$

The change in the probability of experiencing eye irritation due to a change in ozone is given by differentiating Equation (3-6) with respect to 0x. The following is obtained:

$$dp(Ox) = \frac{.0907 * (1-.0407) * (\exp (4.96 - .0907 * Ox)) * dOx}{(1 + \exp (4.96 - .0907 * 0x))^2} \quad (3\text{-}8)$$

Changing the ozone units to parts per million (ppm), and simplifying, Equation (3-8) becomes :

$$\text{Change in daily eye irritation} = 8.7 * \frac{2.718^{(4.96 - 9.1 * 02)}}{[1 + 2.718^{(4.96 - 9.1 * 02)}]^2} * \text{change in O2} \quad (3\text{-}9)$$

where :

02 = daily high ozone hour in ppm.

Upper and lower ranges for eye irritation were not estimated for this analysis.

3.3 DOLLAR VALUE ESTIMATES FOR HEALTH EFFECTS

Dollar value estimates for health effects associated with changes in ozone are based on rough estimates of the value of time relative to a work loss day, which provides a benchmark of the $80 average daily wage in California for 1985. These estimates should be viewed as plausible values, not concrete numbers, because there is very little information available about how individuals would value the time lost for each of these health effects.

RRAD tend to be somewhat less restrictive than the average RAD. The value per RRAD was therefore calculated by assuming that the ratio of days with significant restriction to days with minor restriction is 3.5/19 rather than the 7/19 ratio used for RAD (see Section 2.3). This resulted in a dollar estimate of $31 per RRAD (3.5119 * 80 + 15.5/19 * 20 = 31). Asthma attacks are presumed to imply more serious restrictions in activities than an average RAD, and are given a value of $60, implying that roughly three-quarters of them are equivalent to a work loss day. Days with eye irritation are given a value of $8, one-tenth the daily wage.

3.4 CHANGES IN OZONE FOR DIFFERENT SCENARIOS

The 1984 ozone levels in Santa Clara County are used as the baseline for the calculations of reductions in health effects that would occur if ozone levels in the county were reduced. Two different measures of ozone are needed to calculate the changes in health effects: (1) the two-week average of the daily high hour, and (2) the daily high hour. All three equations are summed over a 36-week period starting March 1. Ozone levels in November through February are typically much lower than during the other months of the year and are unlikely to be associated with any significant health effects.

To calculate the changes in the two measures of ozone, hourly ozone readings were obtained for 1984 for each of the five monitors in the county: (1) Gilroy, (2) Los Gatos, (3) Mountain View, (4) Alum Rock, and (5) San Jose 4th Street. For each of the alternative ozone scenarios, changes in the hourly levels were estimated and the two measures were calculated.

1984 Baseline Ozone

Year-round data were available for the Los Gatos and Alum Rock monitors. At the other monitors, data are not collected during December, January or February. The ozone measures for the 36-week period starting March 1 were not affected by the missing months. When data for single days were missing, the averages were calculated on the basis of the number of days for which data were available. For the daily high, missing days were replaced by the average of the daily high for the 36-week period at that monitor.

Table 3-1 shows the number of days in 1984 on which the Federal or California ozone standards were exceeded at each monitor.

Meeting the Federal Standard

The first scenario was to consider what ozone levels would have been if the Federal standard for hourly ozone were met. The Federal standard is that the maximum hour each day is not to exceed .12 ppm more than once a year. Because

Table 3-1
Number of Days Exceeding Ambient Ozone Standards
in Santa Clara County in 1984

Monitor	Days With One or More Hours Exceeding Federal Standard*	Days With One or More Hours Exceeding California Standard**
Alum Rock	4	19
Gilroy	3	20
Los Gatos	13	30
Mountain View	0	9
San Jose	7	24

* The Federal standard is .12 ppm, not to be exceeded more than once a year on average.
** The California standard is .10 ppm not to be equaled or exceeded.

of the variation in ozone that occurs due to fluctuations in natural conditions, this standard is based on an average over several years rather than for one year at a time. In other words, the standard can be exceeded more than once in a single year if on average it is not exceeded more than once. Since our calculations are based on one year's data, this was simplified to not exceeding .12 ppm more than once in 1984.

To estimate what the levels of ozone would be if the Federal standard were met, the percentage change in the second high value for the year at each station that would bring it to .12 ppm was calculated. These are shown in Table 3-2. It was assumed that every hour would change by the same percentage except that hours at .04 ppm or less were assumed to remain unchanged and no hours were reduced below .04 ppm. The 18 two-week averages were then recalculated.

Meeting the California Standard

The California hourly standard for ozone is .10 ppm, not to be equaled or exceeded. The percentage change that would bring the annual high hourly reading to .09 was calculated for each monitor. It was again presumed that every hour would change by the same percentage if the standard were to be met, except those hours that were at .04 or below. The percentage changes for each monitor are shown in Table 3-2.

Attaining Low Ozone Levels

Based on conversation with ARB staff, .04 ppm was selected as a plausible low level of ozone that is observed at times in areas where there are few manmade emissions of ozone precursors. To calculate the ozone measures for this scenario, every hour exceeding .04 was reduced to .04, and every hour already at .04 or below was left unchanged.

Levels of Ozone Where Health Effects Begin to Occur

Evidence from the health effects literature is not conclusive as to the level of ozone at which health effects begin to occur. For example, Portney and Mullahy

Table 3-2
Percentage Changes in Ozone Levels
To Meet Standards

Station	1984 Second High Hour	% Change to Federal Standard*	1984 Highest Hour	% Change to California Standard**
Alum Rock	.14	14.3%	.15	40.0%
Gilroy	.14	14.3%	.16	43.8%
Mountain View	.11	no change	.12	25.0%
Los Gatos	.16	25%	.17	47.1%
San Jose	.13	7.7%	.16	43.8%

* Second high does not exceed .12 ppm.
** Highest hour does not exceed .09.

found no significant difference between the estimated effect of ozone on RRAD above and below .05 ppm (two-week average of daily high).

We have therefore calculated reductions in health effects using alternative assumptions regarding the level of ozone below which no health effects are expected. The assumptions used for each of the three alternative reductions in health effects are:

Meeting Federal Standards	No Health Effects Below .12 ppm
Meeting California Standards	No Health Effects Below .09 ppm
Attaining Low Ozone Levels	No Health Effects Below .04 ppm

Some ambiguity occurs as to the appropriate way to calculate health effects reductions that would be associated with attaining each of the standards. Due to the unpredictable fluctuations in weather conditions that contribute to the formation and retention of ozone in the atmosphere, it would probably be necessary to reduce emissions of ozone precursors throughout the ozone season in order to bring the highest.levels of ozone down to the standards. This means that levels currently below the standards would also be reduced. The uncertainty is whether there would be any health benefit for reductions in ozone from levels that are already below the standards.

For the scenario involving attainment of the Federal standard we have presumed that there are no health effects below the standard, but to illustrate the effect of this assumption we also calculated the reduction in health effects assuming health effects down to .09 ppm and down to .04 ppm. Table 3-3 illustrates that the effects of this assumption are fairly dramatic. For example, the best estimate of the number of RRAD reduced if the Federal standard is met and there are no health effects below .12 ppm is about 13 thousand. Assuming the Federal standard is met (i.e., using the same percentage reduction in ozone) and that there are health effects as low as .09 ppm, results in an estimate of 45 thousand RRAD reduced. Assuming effects down to .04 ppm results in a reduction estimate of 240 thousand RRAD. The estimates presented in Table 3-3 indicate that the choice of the level of ozone below which no health effects

Table 3-3
Predicted Reductions in Health Effects Associated with Alternative Assumptions

	Expected Number of Cases Reduced (Total County)		
	Eye Irritation (thousands)	Respiratory Restricted Activity Days (thousands)	Asthma Attacks (thousands)
Assuming No Health Effects Below 0.12 ppm			
Meeting Federal Standards			
Lower		11.5	0.1
Best	24.7	13.1	0.4
Upper		14.7	0.7
Assuming No Health Effects Below 0.09 ppm			
Meeting Federal Standards			
Lower		38.5	0.3
Best	70.0	45.1	1.2
Upper		51.7	2.3
Meeting California Standards			
Lower		62.5	0.5
Best	99.0	74.4	1.9
Upper		86.4	3.6
Assuming No Health Effects Below 0.04 ppm			
Meeting Federal Standards			
Lower		190.0	1.2
Best	204.1	240.6	4.6
Upper		291.1	8.6
Meeting California Standards			
Lower		449.9	2.9
Best	418.8	608.8	11.0
Upper		767.7	20.5
Lowest Ozone Level			
Lower		557.1	3.6
Best	472.6	785.8	13.7
Upper		1014.6	25.4

occur has a greater impact on the estimates than does the difference the percentage reductions in ozone required to bring the high levels down to the standards.

3.5 ESTIMATING EXPOSURES OF THE POPULATION TO OZONE

The five ozone monitors are spread throughout the county. In order to estimate exposures to the entire population using the data from the five monitors, census tract groups were formed with three or four adjacent tracts for the more populated parts of the county where the census tracts cover fairly small geographic areas. Distances from the nearest one to three monitors were then measured to the center of each tract group. A judgment was made in each case as to which monitors to include for each tract group, based on the distances and directions to the nearest monitors. An ozone value was then calculated for each tract group by weighting the ozone levels at each of the selected monitors by the inverse of the squared distance from the monitor to the center of the tract group. This gives strongest weight to the nearest monitor.

3.6 RESULTS

The estimated number of cases that would be prevented for each of the potential reductions in ozone is shown in Table 3-4. The estimates for each of the hypothesized ozone reductions assume that there are no health effects below each of the respective levels. The best estimate of the annual reduction in RRAD for the total county that would occur if the Federal standard were met, and if there were no health effects below the Federal standard, implies about 1 RRAD for every 100 people. The reduction in eye irritation cases would be about 1 for every 50 people, and the reduction in asthma attacks would be about 1 for every 250 asthmatics. The best estimate of the annual reduction in RRAD that would occur if the California standard were met, and if there were no health effects below the California standard, implies about 1 RRAD for every 20 people. This is smaller than the reduction in RAD predicted if the California PM standard is met (see Chapter 2). The reductions in eye irritation would be about 1 for

Table 3-4
Predicted Reductions in Health Effects Associated
With Alternative Reductions in Ozone from 1984 Levels

	Number of Cases Reduced		
	Eye Irritation (thousands)	Respiratory Restricted Activity Days (thousands)	Asthma Attacks (thousands)
San Jose			
Meeting Federal Standards			
Lower		3.4	0.0
Best	6.5	4.2	0.1
Upper		4.8	0.2
Meeting California Standards			
Lower		24.6	0.2
Best	36.3	29.7	0.8
Upper		34.7	1.4
Lowest Ozone Level			
Lower		259.8	1.7
Best	219.9	367.0	6.7
Upper		474.1	11.8
Remainder of County			
Meeting Federal Standards			
Lower		7.9	0.1
Best	18.2	8.9	0.3
Upper		9.9	0.5
Meeting California Standards			
Lower		37.9	0.3
Best	62.7	44.8	1.1
Upper		51.6	2.2
Lowest Ozone Level			
Lower		297.3	1.9
Best	252.7	418.9	6.9
Upper		540.5	13.6
Total County			
Meeting Federal Standards			
Lower		11.5	0.1
Best	24.7	13.1	0.4
Upper		14.7	0.7
Meeting California Standards			
Lower		62.5	0.5
Best	99.0	74.4	1.9
Upper		86.4	3.6
Lowest Ozone Level			
Lower		557.1	3.6
Best	472.6	785.8	13.7
Upper		1014.6	25.4

every 15 people. The reductions in the number of asthma attacks would be about 1 for every 50 asthmatics.

Applying the dollar value estimates discussed in Section 3.3 to the number of cases shown in Table 3-4 gives the dollar estimates for predicted changes in health associated with potential reductions in ozone in Santa Clara County. These are shown in Table 3-5. The total best estimate for meeting the Federal standard implies about $.45 per person. About 30% of the county total for meeting the Federal standard falls in San Jose. The total best estimate for meeting the California standard implies about $2 per person for the county as a whole. This is smaller than the per capita dollar estimate for the morbidity changes associated with meeting the California PM standard. About 40% of the county total for meeting the California standard falls in San Jose.

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Table 3-5
Dollar Values for Predicted Reductions in
Health Effects Associated with Ozone
(Thousands of Dollars)

	Dollar Amount Reduced			
	Eye Irritation	Restricted Activity Days	Asthma Attacks	Total Dollar
San Jose				
Meeting Federal Standards				
Lower	51.9	112.5	1.7	166.2
Best	51.9	131.0	7.0	190.0
Upper	51.9	149.5	12.3	213.8
Meeting California Standards				
Lower	290.6	762.9	11.4	1065.0
Best	290.6	919.8	46.3	1256.7
Upper	290.6	1076.7	81.1	1448.4
Lowest Ozone Level				
Lower	1759.1	8054.1	100.1	9913.3
Best	1759.1	11375.5	405.0	13539.5
Upper	1759.1	14696.8	709.8	17165.7
Remainder of County				
Meeting Federal Standards				
Lower	145.4	245.1	4.3	394.8
Best	145.4	276.4	15.7	437.5
Upper	145.4	307.7	30.6	483.8
Meeting California Standards				
Lower	501.5	1175.1	18.8	1695.4
Best	501.5	1387.8	68.3	1957.5
Upper	501.5	1600.4	133.4	2235.2
Lowest Ozone Level				
Lower	2021.6	9215.4	114.7	11351.6
Best	2021.6	12985.7	416.3	15423.6
Upper	2021.6	16756.1	813.2	19590.9
Total County				
Meeting Federal Standards				
Lower	197.4	357.6	6.1	561.0
Best	197.4	407.4	22.7	627.5
Upper	197.4	457.2	42.9	697.5
Meeting California Standards				
Lower	792.1	1938.0	30.2	2760.4
Best	792.1	2307.6	114.5	3214.2
Upper	792.1	2677.1	214.5	3683.7
Lowest Ozone Level				
Lower	3780.7	17269.5	214.7	21264.9
Best	3780.7	24361.2	821.2	28963.1
Upper	3780.7	31452.9	1523.0	36756.6

4.0 THE HEALTH EFFECTS RELATED TO LEAD IN GASOLINE IN SANTA CLARA COUNTY

4.1 INTRODUCTION

Since 1973, EPA has regulated the lead content in gasoline pursuant to Section 211 of the Clean Air Act. Lead has been regulated to both assure the availability of unleaded gasoline for those vehicles with pollution-control devices and to reduce the adverse health effects related to exposure to lead. Lead from leaded gasoline is the largest source of ambient air lead. It enters the ambient air from automobile exhaust and eventually deposits itself on the street, soil, plants, or as household dust. Young children get a particularly large exposure because of pica, the ingestion of nonfood substances such as dust and dirt. Gasoline lead appears to have accounted for 60 percent of the lead in Americans in the second half of the 1970s (Schwartz et al., 1985).

Recent research (Schwartz et al., 1985) has indicated the existence of a strong association between lead in gasoline and the levels of lead in the blood of children and adults. Analyses also indicate that gasoline lead not only increases the average level of blood lead, but also raises the number of children with dangerously high blood lead levels. In addition, the evidence now indicates that increases in blood-lead in adults may have significant health consequences. These realizations have led to the promulgation of an EPA ruling in 1985 requiring further reductions in the amount of lead in gasoline and the consideration of a total ban on leaded gasoline as early as 1988. Implicit in this action is the belief that even in areas where the national ambient air quality standard for lead is being attained, such as in Santa Clara County, additional risks to the public health remain. Thus, it is of interest to determine the extent of health effects related to lead from gasoline in Santa Clara County.

The adverse health effects of high levels of lead in children are well accepted (see EPA, 1986a,b,c; and Schwartz et al., 1985 for reviews). These effects include damage to the kidney, the reproductive system, the liver, blood creation, basic cellular processes, and brain functions. The Centers for

Disease Control (CDC) recommends that children with blood lead levels above 25 micrograms per deciliter (µg/dl) of blood receive follow-up testing and possible medical treatment. Many of these children are then labeled "lead toxic" under CDC criteria.

Increasing evidence also indicates that significant subclinical effects may be occurring at blood lead levels below 25 to 30 µg/dl. These effects include changes in EEG patterns, impairment of heme synthesis, inhibition of certain enzyme activities, impairment of vitamin D activity, and possible interference with neurotransmission. In addition, a number of studies indicate that at levels below 30 µg/dl, lead can affect cognitive functions, as measured by lower IQ tests and other means, as well as cause other neurobehavioral effects in children.

Research has also indicated a number of adverse effects for adults exposed to moderate levels of lead (EPA 1986c). For example, Pirkle et al., 1986, have demonstrated a strong relationship between blood lead levels and blood pressure, with no apparent threshold or "no effects" levels. This finding is significant, since high blood pressure is a major contributor to several cardiovascular diseases.

To estimate the effects that lead emissions from automobiles have on human health, we have employed a three-step process. First, we use existing information to determine the impact of leaded gasoline on the arithmetic mean levels of lead in blood in children and adults. Since it is well documented that blood lead levels are log-normally distributed in the population, with information on the geometric standard deviation we can determine the percent of each group with blood lead exceeding any given level. Next, we used these estimates of blood lead levels to predict the subsequent health effects. This is determined by using dose-response information (with the dose being blood lead levels) from the existing literature. Since many of these studies have identified threshold levels, and since we have estimated the percent of the population with each blood lead level, we incorporated this information into our estimates. Thus, we combined the information on the distribution of blood lead levels with that on the health effects predicted to occur at each level to estimate the total incidence for the population. Since many different sources

of lead exposure exist, the final step is to take this total and determine the amount that is a result of air emissions.

4.2 ESTIMATING THE EFFECT OF GASOLINE USAGE ON BLOOD LEAD

When leaded gasoline is burned in an engine, small amounts of lead are deposited in the engine and exhaust system, but most of it is emitted from the tailpipe to the air, where it ultimately settles to the ground. People are exposed to lead from gasoline through a variety of routes, including direct inhalation of lead particles, ingestion of lead-contaminated dust or inhalation of such dust when it is stirred up, and ingestion of food or water that has been contaminated with lead. Although it is difficult to model the contribution of each of these separate pathways, several studies have demonstrated a strong and consistent relationship between the usage of gasoline lead and subsequent mean blood lead.

In its regulatory analysis of the 1985 rulemaking further reducing the lead content in gasoline, EPA used data from the Second National Health and Nutrition Examination Survey (NHANES II) to estimate the relationship between gasoline lead and blood lead over time (Schwartz et al., 1985). Conducted between 1976 and 1980, NHANES II contains socioeconomic and health information for over 27,000 individuals from 64 sampling areas throughout the U.S. From this population, blood samples from a subset of 10,000 people were collected under rigorous specifications. Leaded gasoline usage was determined by combining monthly data on leaded gasoline sales (from the U.S. Department of Energy) with quarterly average lead concentrations in grams per gallon (reported to EPA by refiners). Arithmetic mean blood lead levels for the cities sampled in 1976 by NHANES II are shown in Table 4-1. San Jose is one of these cities, and the figures in Table 4-1 suggest that levels in San Jose are close to average for that year.

Multiple regression analysis was used to relate mean blood levels for the entire NHANES II sample, 1976-1980, to gasoline lead for children and adult males and females. The analysis specifically controlled for the independent influence of age, race, sex, income, degree of urbanization, region of the country,

Table 4-1

Mean Blood Lead Levels from NHANES II

for Cities Sampled in 1976

	Arithmetic Mean Blood Lead (µg/dl)*			
	Children Under 6	Children 6 to 17	Men 18 and Over	Women 18 and Over
San Jose, CA	18.3 (23)	14.8 (19)	16.6 (20)	12.7 (18)
Fresno, CA	20.9 (84)	14.6 (50)	15.7 (66)	12.6 (58)
Highland Park, IL	17.6 (26)	14.7 (26)	22.7 (29)	17.1 (24)
Des Moines, IO	22.3 (35)	15.8 (20)	19.7 (31)	13.4 (39)
Shreveport, LA	25.2 (39)	15.4 (41)	19.3 (37)	13.8 (58)
Minneapolis, MN	16.5 (83)	10.7 (53)	18.9 (81)	14.1 (76)
Greenville, SC	20.2 (63)	15.1 (34)	17.0 (53)	11.8 (72)
Newport News, VA	22.1 (45)	13.5 (31)	20.5 (35)	15.7 (36)
Tacoma, WA	19.3 (40)	16.2 (24)	21.8 (35)	15.8 (25)
MEAN OF CITY MEANS for 1976	20.3	14.5	19.1	14.1

* Number of observations in parentheses.

These are NHANES II locations with populations exceeding 100,000. In each case, the sample area covered the entire county, not just the city.

educational level, smoking habits, alcohol consumption, occupational exposure, dietary factors, and interactions among some of these factors.

The results proved very consistent and robust over a number of alternative regression specifications and functional forms. The regressions indicated that gasoline lead usage has a positive and significant effect ($p < 0.0001$) on subsequent blood lead. The effect was very similar across all of the subgroups (e.g., children, teens, males, females, urban, rural) and indicates that a national increase of lead in gasoline of 100 metric tons per day, lagged one month, results in a 2.14 µg/dl increase in mean blood lead.

Since the NHANES II included San Jose in their survey in the first quarter of 1976, we can use the actual measured blood lead levels in our estimate. However, we must adjust this estimate to determine blood lead levels in 1985 since the amount of lead used in gasoline fell dramatically during this time interval. Data suggest that lead usage in the U.S. dropped from 443.5 metric tons per day in the first quarter of 1976 to approximately 68.5 metric tons per day during 1985, a decrease of 85 percent. Ambient air quality data for San Jose indicated a similar 85 percent decrease in the annual average concentration of lead in the air during this period. We also estimated relevant decreases in lead for all of California and for San Jose. Based on data from the California Air Resources Board, Compliance Division; the Ethyl Corporation; and the U.S. EPA, Office of Mobile Sources, Refinery Data, we estimated a 91.5 percent decrease in lead emissions in California, and a 91.2 percent decrease in San Jose from 1976 to 1985. We would expect a greater decrease in lead in California relative to the entire nation since California had an interim lead standard of 0.8 gm/gallon in effect as of September 1984 (while the national standard was 1.1 gm/gallon).

Therefore, we can convert the California data into a national equivalent and can calculate the 1985 arithmetic mean blood lead level for subgroup i (BLi) as:

$$1985\ BLi = 1976\ BLi - 2.14 \times (\text{change in gas lead in hundreds of metric tons per day})$$

As displayed in Table 4-1, the NHANES II data indicated that in San Jose, children less than six years of age had an arithmetic mean blood lead level of 18.35 µg/dl. Individuals age 6 to 17 had an arithmetic mean blood level of 14.84, adult males had an average of 16.60, while adult females had an average of 12.67. Using the above equation, the estimated 1985 mean blood lead levels for children less than 6, individuals 6 to 17, adult males, and adult females are 9.65, 6.15, 7.95 and 4.0, respectively.

It is also necessary to adjust the 1985 baseline level to account for other reductions in lead that have occurred between 1976 and 1985. Besides the direct inhalation and ingestion of gasoline lead, the greatest source of change in lead intake during this period is probably due to reductions in dietary lead. There are two main sources of dietary lead: (1) air lead that is deposited onto vegetation, soils and water; and (2) the processing, transportation, packaging, and storage of food during which lead can be introduced either through atmospheric deposition, or through metallic contamination, particularly from solder.

To calculate the change in blood lead resulting from this first factor, we weighted the proportion of total lead intake due to indirect deposition of lead (based on U.S. EPA, 1986b) by the reduction in gasoline lead that occurred during 1976-1985. This assumes that the lead content of vegetation, soils and water responds reasonably quickly to changes in leaded gasoline. In fact, evidence suggests that lead many continue to exist in soil well after gasoline lead is no longer used. Thus, we are making an assumption that the benefit stream, which will actually occur over many years and decades, will occur at the time of the reduction in lead. This simplification will result in an over-estimation of the health benefits of eliminating gasoline lead in year one.

To calculate the reduction due to lead solder, we incorporated information (U.S. EPA, 1986b) on the contribution of lead solder to blood lead levels in the 1970s, and estimations of the reductions in the use of solder since that time.

These two dietary factors combined indicate that children below age 6 may have had an additional reduction in blood lead of 2 µg/dl. The other three groups likely will have had reductions of around 1.5 µg/dl. The difference is largely

a result of the voluntary reduction by metal can manufacturers of lead solder used for packaging baby food.

We assume that there have been zero or only negligible changes in lead exposure from other sources including lead from paint, drinking water, point sources, and occupational exposure, an assumption supported by discussions with local health officials. Thus, the estimated 1985 baseline levels for lead for children less than 6, individuals 6 to 17, adult males, and adult females are 7.65, 4.65, 6.45 and 2.50, respectively.

Once the 1985 mean levels for children and adults are established, we can use the log-normality assumption about the distribution of blood lead levels to determine the percent of the population at each level. Based on Schwartz et al. (1985), we calculated the relationship between the geometric standard deviation (GEOSD) and the arithmetic mean (AMEAN) for children below age 6, and for all others in the population. The equations are:

Children < 6:

$$\underset{(.034)}{GEOSD = 2.058} \; \underset{(.003)}{- 0.041 * AMEAN}$$

All Others:

$$\underset{(.049)}{GEOSD = 1.933} \; \underset{(.005)}{- 0.044 * AMEAN}$$

Standard errors of the estimated coefficients are in parentheses and indicate that the coefficients are statistically significant at the 95% confidence level. These equations indicate that a shift in the mean blood lead due to a change in gasoline lead will not result in equal absolute reductions in the distribution of lead levels across the population. Because of exposure to other sources of lead, some individuals will continue to have high blood lead, even as exposure to gasoline lead decreases.

To estimate the health effects attributed purely to air lead, we first calculated the health effects associated with this 1985 mean blood level. Then, we subtract from these estimates, the health effects associated with the blood lead that would result with a zero level of lead in gasoline. The difference is the health effects attributable to 1985 gasoline lead levels. The procedure necessitates a calculation of the expected mean blood lead levels associated with zero lead in gasoline. We used the same procedure as above to estimate these mean blood lead levels. We calculated the decline in blood lead due to reductions in both the inhalation and ingestion of lead, and the indirect intake through the diet. These factors imply a reduction in the 1985 baseline level by 0.8 and 0.1 μg/dl, respectively. Thus, the mean blood lead levels that are estimated to exist at zero gasoline lead would be 6.75 μg/dl for age less than 6, 3.75 for age 6 to 17, 5.55 for male adults, and 2.0 for female adults (assumed background level).

4.3 ESTIMATING HEALTH EFFECTS RELATED TO LEAD

As noted earlier, there is extensive scientific evidence documenting the health effects associated with lead exposure in both children and adults. To estimate the risk to public health at different exposure levels, we rely on dose-response curves that relate exposure (measured as blood lead levels) to incidence of an effect in the exposed population. Unfortunately, despite the vast amount of documented effects of lead, only a few studies actually provide the information necessary to calculate dose-response curves. For this reason, we can only estimate the potential risks for a subset of the health effects including blood effects, fetal developmental effects, reproductive effects in men, renal effects, neurological effects, and cardiovascular effects in men (for a complete review of the effects of lead see Schwartz et al, 1985 and U.S. EPA, 1986a, 1986c). We have also not incorporated very recent information including the effects of lead on stature (Schwartz et al., 1986; U.S. EPA, 1986c).

There are two other limitations inherent in our estimates. First, for several of the organ systems, increasing lead levels are associated with increasing severity of effect. However, the dose-response curves we used can only predict a single effect and it is therefore assumed that increasing exposure is

associated only with increasing incidence of the particular effect. This limitation is less apparent when data are available to estimate two different effects for a particular organ system. This is the case in our estimates of hematological effects, where data were available to calculate the risk for anemia (a potentially severe effect) and for increased levels of free erythrocyte protoporphyrin (a milder effect indicative of a disturbance in hemoglobin synthesis).

Another limitation involves the use of blood lead levels as a measure of cumulative exposure. Although blood lead is not a good indicator of cumulative exposure, it is used as such in the majority of human research conducted to date. It is probably a more useful marker of current exposure. Nevertheless, blood lead is the standard approach used for lead exposure screening and evaluation.

In the section that follows, we will identify the study that is used to generate dose-response estimates for each of the health effects for which we will provide risk estimates. We will not attempt to review all of the existing literature on that health effect. This next section relies heavily on Perlin (1986).

Blood Effects

The hematological effects of lead are well understood and there is information on the biochemical mechanisms and the dose-effect and dose-response relationships. There are two types of effects. Lead interferes with heme and hemoglobin synthesis, and affects the erythrocyte's (red blood cell) morphology, function and lifespan. The clinical manifestation of these effects is anemia. Basically, lead can interfere with many of the steps in the biosynthetic pathway that produces hemoglobin. With increasing lead exposure, some of the intermediate products of this process, such as free erythrocyte protoporphyrin (FEP), are disturbed and will begin to appear in the blood or urine. continued disruption of this process can lead to anemia, and can lead to adverse effects on many other physiological processes.

Separate dose-response curves have been calculated for adults and children based on the increase in FEP in the blood versus blood lead. Although there is

controversy over the clinical significance of small increases in FEP (or any of the other intermediate products), these dose-response curves indicate the proportion of exposed individuals having some lead-induced disturbance in the hemoglobin synthesis. The curve for children is based on data on children living various distances from a smelter (Roels et al., 1976). The curves for adults are based on "moderately" lead-exposed workers and nonexposed controls (Roels et al., 1975).

With continued exposure to lead, the blood-forming processes can be severely disrupted so that the individual develops anemia, clinically diagnosed as a hemoglobin concentration less than 9.5-11.0 µg/dl blood. A dose-response curve for children is based on the data of Betts et al. (1973). Dose-response information for adults was not available so anemia cases were not estimated for this group.

Fetal Developmental Effects

Studies have shown that exposure to lead during pregnancy can lead to abnormalities in their children, such as growth retardation and neurological damage, and may be a factor contributing to mental retardation. For example, Bellinger et al. (1986) found an association between umbilical cord blood lead and mental development in middle class Boston children. Dietrich et al. (1986), in an analysis of inner city children in Cincinnati, found an association between blood lead and lowered performance on tests of mental development, psychomotor development, and behavior. Further, they reported a significant negative association between prenatal lead exposure and gestational age. The dose-response curve was based on the data of Needleman et al. (1984), who examined 5,183 neonates within the first two days after birth and found a statistical relationship between umbilical cord blood lead levels and the incidence of minor congenital anomalies such as hemangiomas and lymphangiomas, hydrocele, minor skin anomalies, and undescended testicles. Although these effects were. classified as "minor anomalies" they may be important markers of impaired development. This study was not designed to measure the impact of lead on the more serious health outcomes.

Since the exposure data were expressed in terms of mean umbilical cord blood lead levels, the study of Fahim et al., (1975) was used to convert these into estimates of maternal blood lead levels. Thus, the resulting dose-response curve relates estimated maternal blood lead levels to the risk of bearing a child with minor anomalies.

Reproductive Effects

Studies have identified reproductive problems in both men and women exposed to lead (Rom, 1980). Many effects have been noted in occupationally exposed men, including subnormal sperm counts, abnormal sperm morphology and/or motility, and lower chromosome stability. Effects noted in women (exposed directly or affected through the lead exposed husband) include abnormal pregnancies (spontaneous abortion, stillbirth or early neonatal loss), menstrual disorders, and infertility.

Unfortunately, appropriate data were found only to estimate a dose-response relationship for reproductive effects in men. The curve was based on the study of Lancranjan et al. (1975) in which 150 men from a lead storage battery plant were examined for several indices of reproductive effects. This study found a statistically significant dose-response relationship between the incidence of abnormal sperm motility, density and morphology and mean blood levels. This health effect should not be viewed as the incidence of male infertility but rather the incidence of some lead-induced effect on the reproductive system.

Renal Effects

Lead exposure in children, adults, and test animals has been associated with physiological and morphological changes in the kidney. At low doses, lead is associated with the interference of cellular functions in the kidney, including energy metabolism, respiration, oxidative phosphorylation, and synthesis of heme, proteins, and vitamin D hormone. A dose-response curve for renal effects in adults has been estimated based on the findings in two occupational studies: Wedeen et al. (1979) and Lillis et al. (1968).

In the Wedeen et al. study, renal disease was defined as reduced glomerular filtration rate and in the Lillis et al. study, it was identified by reduced urea clearance. Each study ruled out the possibility of kidney problems resulting from causes other than lead exposure. Thus, the dose-response curve for adults relates the incidence of renal dysfunction versus mean blood levels.

For children, a dose-response curve was estimated based on lead's interference with vitamin D metabolism, much of which is controlled by the kidney. Given the complicated nature of the vitamin D metabolism, no single dose-response curve can adequately describe the relationship between lead exposure and effects on this system. Thus, the curve used here is a simplification of the situation and merely relates group mean blood levels to the levels of serum (1,25-(OH)2D), the major active form of vitamin D. Therefore, the dose-response curve represents effects on the kidney only to the extent that the serum level of active vitamin D can be used as an indicator of lead's interference with normal renal function.

Neurobehavioral Effects

Lead can induce both structural and functional effects on the nervous system and can affect the motor and sensory nerves. The effects of lead can result in deterioration of intellectual, sensory, neuromuscular, and/or psychological function in both children and adults. Several studies have demonstrated alterations in brain functions at relatively low exposure levels, particularly when exposure occurs during early development of the individual. There are numerous epidemiological and clinical studies concerning the neurobehavioral effects of lead in both children and adults. Controversy remains, however, over the adequacy of the study designs, the adequacy of the control of potential confounders, and the interpretation of the test results.

The dose-response curve for neurobehavioral effects in adults is based on the study of Seppalainen et al. (1979), which examined motor nerve conduction velocity in individuals occupationally exposed to lead. The velocity provides an indication of neurophysiological function in both sensory and motor nerves (the peripheral nervous system). The dose-response curve represents the proportion of individuals having slowed motor conduction velocities in one or more nerves versus the time-weighted average blood lead levels.

Similar to the situation in adults, substantial evidence indicates that lead exposure in children can induce neurological dysfunction. This issue has attracted much attention and generated considerable controversy for many years due to the conflicting results of numerous studies conducted to examine neurotoxic effects in children exposed to relatively low lead levels.

In contrast to the dose-response curves for adults, which is based on effects on the peripheral nervous system, the dose-response for young children is based on effects on the central nervous system (i.e., the brain and spinal cord). Decrements in IQ score have been used as the critical neurotoxic effect in children. Based on data from Schwartz (1986) and Schwartz et al. (1985), the curve is calculated by assuming that approximately one-third of the children with blood lead levels of 25 µg/dl or greater would have a decrease in IQ of at least 2-3 points. This assumption is based on information suggesting an IQ decrement of 1-2 points for blood lead levels of 10-25 µg/dl, of 2-3 points for 25-40 µg/dl and 4-5 points for 40-60 µg/dl.

Cardiovascular Effects

Studies in both humans and experimental animals indicate that lead exposure is associated with a variety of effects on the cardiovascular system. Hypertension, or increased blood pressure, represents the most significant effect associated with lead exposure. For example, Pocock et al. (1985) found an association between blood lead levels and both systolic and diastolic blood pressure. Relationships between blood lead and blood pressure among American adults have recently been evaluated by Harlan et al. (1985) and Pirkle et al. (1985). These analyses were based on evaluation of the NHANES II data, which provide carefully tested information on blood pressure as well as information on a variety of potentially confounding factors for a representative sample of the U.S. population. As such, these studies avoided the problem of the healthy-worker effect, workplace exposures to other toxic agents, selection bias, and problems of control group selection which often complicate other studies. The analysis of Harlan et al. demonstrated statistically significant linear associations ($p < 0.001$) between blood lead concentrations and both systolic and diastolic blood pressure among males and females, age 12 to 74 years. However,

using a model controlling for a number of potentially confounding factors, blood lead was found to not be independently related to blood pressure in women.

Further analyses reported by Pirkle et al., focused on white males, age 40 to 59 years. In the subgroup studied, significant associations were found between blood lead and blood pressure even after controlling for all risk factors known to be correlated with blood pressure. Therefore, the dose-response curve for lead-induced cardiovascular effects has been based on the association of blood lead level versus incidence of hypertension among adult white males and is calculated only for the subgroup of white males age 40 to 59.

The increase in blood pressure related to an increase in blood lead levels also can lead to a higher incidence of more serious health outcomes, including myocardial infarctions (heart attacks), and death. A number of large scale epidemiological studies including the Pooling Project Research Group (1978) and the Framingham studies (McGee and Gordon, 1976) have shown that elevated blood pressure increases the risk of cardiovascular disease. We will provide estimates, therefore, of these outcomes for the subgroup of males age 40 to 59. We assume that the other age groups are not impacted and therefore probably underestimate these effects.

The Pooling Project pooled the results of five longitudinal studies on the incidence of coronary heart disease (CHD) in middle aged white men. The first incidence of CHD, defined as fatal or nonfatal myocardial infarction and sudden CHD death, was measured over a 10 year period. Using logistic analysis, the research indicated that smoking, serum cholesterol, and diastolic blood pressure were major risk factors in the incidence of CHD.

The Framingham study (McGee and Gordon, 1976) was one of the studies included in the Pooling Project. Besides estimating the incidence of CHD, this study of white middle aged men considered the incidence of deaths from all causes. As a result, diastolic blood pressure was identified as a significant predictor of deaths.

4.4 ESTIMATION OF HEALTH EFFECTS

To estimate the cardiovascular effects related to lead in gasoline we use dose-response information provided by Schwartz (1986) based on the findings of Pirkle et a1.(1985), the Pooling Project and the Framingham studies. Additional documentation for this estimation process is found in Brennan et al (1986). The curve is based on a logistic regression of the probability of hypertension, defined as diastolic blood pressure greater than 90 mm Hg., versus blood lead. The health effects are estimated for the population of adult males age 40 to 59. We determined how the probability of hypertension changes when we move from the baseline of blood lead levels related to 1985 lead in gasoline to blood lead levels associated with the total elimination of lead in gasoline.

To estimate the effect of blood lead on the probability of hypertension, we use the mean values of the NHANES II sample for covariates such as body mass, albumin, hemoglobin, Vitamin C, dietary potassium, total carbohydrates, and recreational. exercise. We then can predict the probability of hypertension as a function of log of blood lead by:

$$\text{Change in H} = (1 + \exp -(-2.744 + 0.793\ (\ln \text{PbB1}))^{-1} - (1 + \exp -(-2.744 + 0.793\ (\ln \text{PbB2}))^{-1}$$

where:

H = the probability of hypertension
PbB1 = old blood lead level (µg/dl)
PbB2 = new blood lead level (µg/dl)

Since we do not have separate estimates of the average blood lead levels for male adults ages 40 to 59, we use our estimate for all males in San Jose in 1985 of 6.45 µg/dl. The new level, if there were no lead in gasoline, was estimated at 5.55 µg/dl. Thus, the equation suggests that for the 119,000 males between age 40 and 59, there will be decrease in the probability of hypertension of 2.0 percent. Thus, our estimates indicate that the lead in gasoline appears to be responsible for 2,380 cases of hypertension in 1985.

Hypertension is believed to contribute to the risks of deaths due to all causes and of heart attacks (both fatal and nonfatal). Since the probability of deaths from all causes and of heart attacks have been estimated as a function of diastolic blood pressure, we first need to find the association between blood lead and diastolic blood pressure. Based on Pirkle et al. (1985) and U.S. EPA (1986c), we use the following relationship:

$$\text{change in DBP} = 2.74\,(\ln \text{PbB1} - \ln \text{PbB2})$$

where:

DBP = diastolic blood pressure.

For the decline in blood lead estimated above, this equation predicts that DBP will decrease by 0.41 mm Hg moving from 1985 baseline levels to a "no lead" scenario.

Using the sample means of the independent variables in the Pooling Project, we can express the relationship between the change in blood pressure and the change in the probability of a CHD event in the following ten years as:

$$\text{change in Pr(CHD)} = (1 + \exp -(-4.996 + 0.030365(\text{DBP1})))^{-1} - (1 + \exp -(-4.996 + 0.030365(\text{DBP2})))^{-1}$$

where:

Pr(CHD) = the 10-year probability of a coronary heart disease event.

DBP1 = old diastolic blood pressure

DBP2 = new diastolic blood pressure

The mean DBP for whites in California in 1979 was 76 mm Hg. (California Department of Health Services, 1982). Assuming a lead-induced decline in DBP of 0.41, as predicted above, the change in the probability of a CHD event in 10 years is 0.00074. For the subgroup of males age 40 to 59, therefore, we estimate that 1985 levels of lead in gasoline are responsble for an average of 8.8 heart attacks per year for the next 10 years.

The Framingham study (Shurtleff, 1974) can be used to estimate the change in mortality due to the change in diastolic blood pressure. Controlling for serum cholesterol levels and smoking, the association can be estimated by:

$$\text{change in Pr(MORT)} = (1 + \exp -(-5.3158 + 0.03516(\text{DBP1})))^{-1} \\ (1 + \exp -(-5.3158 + 0.03516(\text{DBP2})))^{-1}$$

where:

Pr(MORT) = the 12 year probability of death from all causes.

The change in diastolic blood pressure associated with the difference between 1985 gasoline lead levels and zero gasoline lead results in a change in the 12 year probability of death of 0.000887. Thus, for the subgroup of males age 40 to 59, we estimate that the 1985 gasoline lead levels result in 8.8 deaths per year for the next 12 years. This estimate includes deaths due to all causes including heart attacks.

A different methodology is used to calculate health effects that are not cardiovascular-related. To determine the expected number of health effects, we multiplied the estimated health risk at each blood lead level by the proportion of the population at each of the respective blood lead levels. Perlin (1986) tabulated this information which enabled us to determine the ultimate health risk to the population. This procedure incorporates the information that the studies provide about the threshold level for each health effect and the increase in risk as blood lead level increase. The thresholds in µg/dl that are used for each health effect are displayed in Table 4-2. It should be noted that these thresholds have been consistently falling as the methods for detection improve.

We can calculate the total number of health effects at 1985 blood lead levels by using the group risks (the total distribution of risk at each blood lead level as a function of a given mean blood level) calculated by Perlin (1986) and estimates of the portion of the San Jose population in each group based on the 1980 Census. Table 4-3 summarizes the information on the group risk, the population group, and the total number of health effects estimated for 1985. As

Table 4-2

Thresholds Used in Predictions

	Men	Women	Children
Blood/FEP	16.8	13.8	8.2
Reproductive	23	NA	NA
Fetal	NA	13.4	NA
Renal	40	40	18
Neurological	11	11	*
Anemia	NA	NA	36
Hypertension	*	NA	NA
1978 Recommended OSHA Level (1)	<=30	<=30	NA
Proposed EPA 1985 Health Advisory (2)	15	15	15
1985 CDC Guideline for Medical Attention (3)	NA	NA	25

NOTES:
* Calculation presumes no threshold below which no effects are observed.
NA = not applicable

(1) The Occupational Safety and Health Administration, in 1978, recommended that couples wanting to have children should maintain their blood lead levels at or below 30 µg/dl in order to reduce the risk of reproductive effects and/or effects on the fetus. More recent data indicates that this level may not offer enough protection.

(2) The proposed 1985 EPA long-term health advisory concluded that a blood lead level of 15 µg/dl represented a no observed adverse effects level for both children and adults and that higher blood lead levels may be cause for concern. The same source recommended that women of childbearing age maintain their blood lead levels below 15-20 µg/dl in order to reduce the risk of adverse effects during pregnancy.

(3) The CDC recently (1985) revised its guidelines for identifying a child as requiring immediate medical and environmental intervention due to excessive lead exposure. The revised standard is 25 µg/dl blood lead accompanied by a blood erythrocyte protoporphyrin level >= 35 µg/dl.

Table 4-3
Total Health Effects Due to Lead and Other Causes
at 1985 Blood Lead Levels

	Men	Women	Children Age 6-17	Children Age < 6
Population	468,452	468,452	251,037	107,131
1985 Mean Blood Lead (µg/dl)	6.45	2.50	4.65	7.65
Group Risk (per person):				
Blood/FEP	8.11 E-4	5.27 E-5	2.23 E-4	3.17 E-2
Fetal	NA	1.09 E-5	NA	NA
Reproductive	9.07 E-5	NA	NA	NA
Renal	3.45 E-6	2.34 E-8	9.38 E-7	2.64 E-3
Neurological	3.03 E-6	6.57 E-5	9.44 E-4	7.11 E-3
Anemia	NA	NA	0	1.17 E-4
Annual Cases:				
Blood/FEP	380	25	56	3396
Fetal[a]	NA	4	NA	NA
Reproductive[b]	36	NA	NA	NA
Renal	2	0.01	0.2	283
Neurological	1419	31	238	764
Anemia	NA	NA	0	13

NOTES:
* The consequences of a given effect may be different depending on the group. See the text for a full description of the health effect.
NA = Not available or applicable
[a] based on a subset of 337,123 women aged 15-44, bearing 67.4 births per thousand
[b] based on a subset of 396,614 men aged 18-54

an example, consider the effect labeled blood/FEP, a mild hematological effect that indicates that lead is disturbing the process that makes red blood cells. Each of the groups has a different risk estimate because each has a different mean blood lead and dose-response relationship. For example, for adult males, the risk is 0.000811 per person, with a population of 468,452. Thus, there will be 380 expected cases where the blood-forming cycle is disturbed.

Table 4-3 depicts the total effects associated with a certain level of lead in the blood. However, what we really want to determine are the effects that can be attributed to gasoline lead emitted into the air. This is calculated by extrapolating from the gas lead - blood lead relationship down to the level where gas lead is zero. This calculation assumes that the gasoline lead - blood lead relationship continues beyond the range of the original data from which it was estimated. We then can calculate the risks and cases that occur at this zero baseline (i.e., risks that are associated with a zero level of gasoline lead) for each of the groups, and subtract them from the total number of cases, calculated earlier, in Table 4-3. The blood lead that occurs at zero level of gasoline lead can be attributed to many other potential sources including lead in drinking water, from cans with lead solder, from fruits and vegetables that have had lead deposited on them, from lead paint, and from reentrained road, soil and house dust.

The health effects that can be attributed to 1985 gasoline lead in San Jose are displayed in Table 4-4. We make an addition to the estimates of health effects for adult males by using research (Pirkle et al., 1985) that relates the impacts of gasoline lead to hypertension and to a subsequent increase in heart attacks and deaths due to all causes.

The significant effects of lead in the environment can be readily observed, even as California complies with the required reductions in the amount of lead in gasoline. The estimates suggest that in 1985 adult males suffered an additional 165 blood effects (disruption of the process that makes red blood cells), 16 reproductive effects (malformed sperm), 0.07 more renal effects (impaired kidney function), 571 neurological effects (effects on the peripheral nervous system), 2380 additional cases of hypertension, 8.8 fatal and nonfatal heart attacks, and 8.8 deaths from all causes. Also, of particular note, is the large incidence of

Table 4-4

Health Effects Attributable to 1985 Gasoline Lead

	Men	Women	Children Age 6-17	Children Age < 6
1985 Mean Blood Lead with Zero Gasoline Lead (µg/dl)	5.55	2.00	3.75	6.75
Annual Cases:				
Blood/FEP	165	17	34	974
Fetal	NA	2	NA	NA
Reproductive	16	NA	NA	NA
Renal	0.7	0	0.16	95
Neurological	571	20	136	260
Anemia	NA	NA	NA	4
Hypertension[a]	2380	NA	NA	NA
Heart Attacks[b]	8.8	NA	NA	NA
Deaths[c]	8.8	NA	NA	NA

NOTES:

[a] based on 119,000 men aged 40-59.

[b] predicted number of fatal and nonfatal myocardial infarctions related to increases in blood pressure, males ages 40-59.

[c] predicted number of deaths from all causes related to increases in blood pressure, males ages 40-59.

certain effects on children under age six. For example, the estimates suggest that as a result of gasoline lead exposure in 1985, there were 974 blood effects, 95 renal effects (interference with vitamin D metabolism), and 260 neurological effects (impacts on the central nervous system based on problems with mental processes, such as lowered IQ scores).

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5.0 HEALTH EFFECTS OF CARBON MONOXIDE

5.1 INTRODUCTION

When carbon monoxide (CO) enters the respiratory system, it attaches to hemoglobin and forms carboxyhemoglobin (COHb), reducing the oxygen carrying capacity of the blood. CO's affinity for hemoglobin is much greater than that of oxygen and when present in significant amounts can result in a significant reduction in oxygen carried to the tissues of the body. Exposures to very high levels of CO for some period of time (such as in a closed garage with a running automobile) can result in death.

Some population groups are believed to be at higher risk of ill effects of exposures to ambient CO levels that occur in urban areas. These include individuals with heart disease, with chronic respiratory disease, with chronic anemia, and fetuses. The normal healthy population is also at risk of some ill effects (primarily of a less serious nature) at ambient CO levels that do occur in Santa Clara County. In general, the available quantitative information regarding dose-response relationships between CO or COHb levels and adverse health effects is limited. The literature suggests COHb levels above which certain adverse health effects begin to occur in some population groups, but does not allow an estimation of the number of people expected to experience the adverse effects at different COHb levels.

This chapter presents available information about levels of COHb and associated health risks that occur in Santa Clara County. The information on COHb levels presented in this chapter is based on the NHANES II study, conducted in Santa Clara County in 1976. Quantitative estimates of the benefits of meeting Federal or California standards for CO are not presented due to difficulties in determining how COHb levels would change with changes in ambient CO levels measured at stationary monitors.

Exposures to CO are difficult to estimate because CO dissipates rapidly in the environment. CO levels can be quite high near a source (such as a highway) and be quite low a short distance away, reducing the accuracy of using

stationary monitors to measure the exposure of individuals in a geographic area. A detailed analysis could be done using the CO exposure models that have been and are being develped at EPA and elsewhere, but this would have required far more resources than were available for this analysis and would still be subject to considerable uncertainty.

5.2 COHb LEVELS IN SANTA CLARA COUNTY IN 1976

Information on COHb levels in Santa Clara County is available from the National Center for Health Statistics Second National Health and Nutrition Examination Survey (NHANES II), which was conducted throughout the U.S. in 1976 through 1980. A subsample of the respondents to this survey was tested for COHb levels. The survey was conducted in Santa Clara County in February and March 1976.

Reported COHb data for Santa Clara County and the remainder of the survey locations were used to calculate a geometric mean and geometric standard deviation of COHb levels in Santa Clara County. Since smoking has a significant effect on COHb levels, separate estimates were made for smokers and nonsmokers. Former smokers were grouped with never smokers in the nonsmoker group because their COHb levels were quite similar to never smokers, while current smokers had much higher levels. The following COHb means demonstrate the similarity between never smokers and former smokers in terms of current COHb.

COHb Means (Weighted) for the U.S. from NHANES II

	Geometric	Arithmetic
Never Smokers	.7%	.9%
Former Smokers	.8%	1.2%
Current Smokers	3.8%	4.6%

Figures 5-1 and 5-2 show the distributions of COHb levels for smokers and nonsmokers in the NHANES II sample for the U.S. Note the difference in scales on the horizontal axis. The shapes of these distributions look like they can be reasonably approximated by a log-normal distribution.

Table 5-1 shows the calculated COHb means and standard deviations using the NHANES II data. The means in the first two rows are for the 44 locations in the sample with populations exceeding 100,000. This subsample represents about two-thirds of the NHANES II subjects tested for COHb and was selected because it is likely to be more representative for individuals living in or near an urban area.

The weighted means are calculated using the weights included with the data because the stratified sample for NHANES II was not entirely representative of the U.S. population due to the planned over-sampling of individuals in some specific demographic groups. As shown in the table, the weights do not significantly affect the means for the U.S. The only mean that is changed by the weights is for smokers in Santa Clara County, but this is based on only 15 individuals.

The standard deviations presented in the second column are the averages of the geometric standard deviations calculated for smokers and nonsmokers in each NHANES II location with population exceeding 100,000. Since the desired statistic for this analysis is a geometric standard deviation within one location, this average of location-specific deviations was judged preferable to the standard deviation across the whole sample. The latter would reflect variations across different locations as well as the variation within any one location.

The last column shows the means and standard deviations selected for use in this analysis for Santa Clara County. The U.S. mean for smokers was selected over the Santa Clara calculated mean because the latter was based on only 15 individuals and could be unrepresentative if the smoking levels of these 15 people are not typical of all smokers in the county.

Figure 5-1

Frequency Distribution of COHb (%) For Nonsmokers in NHANES II

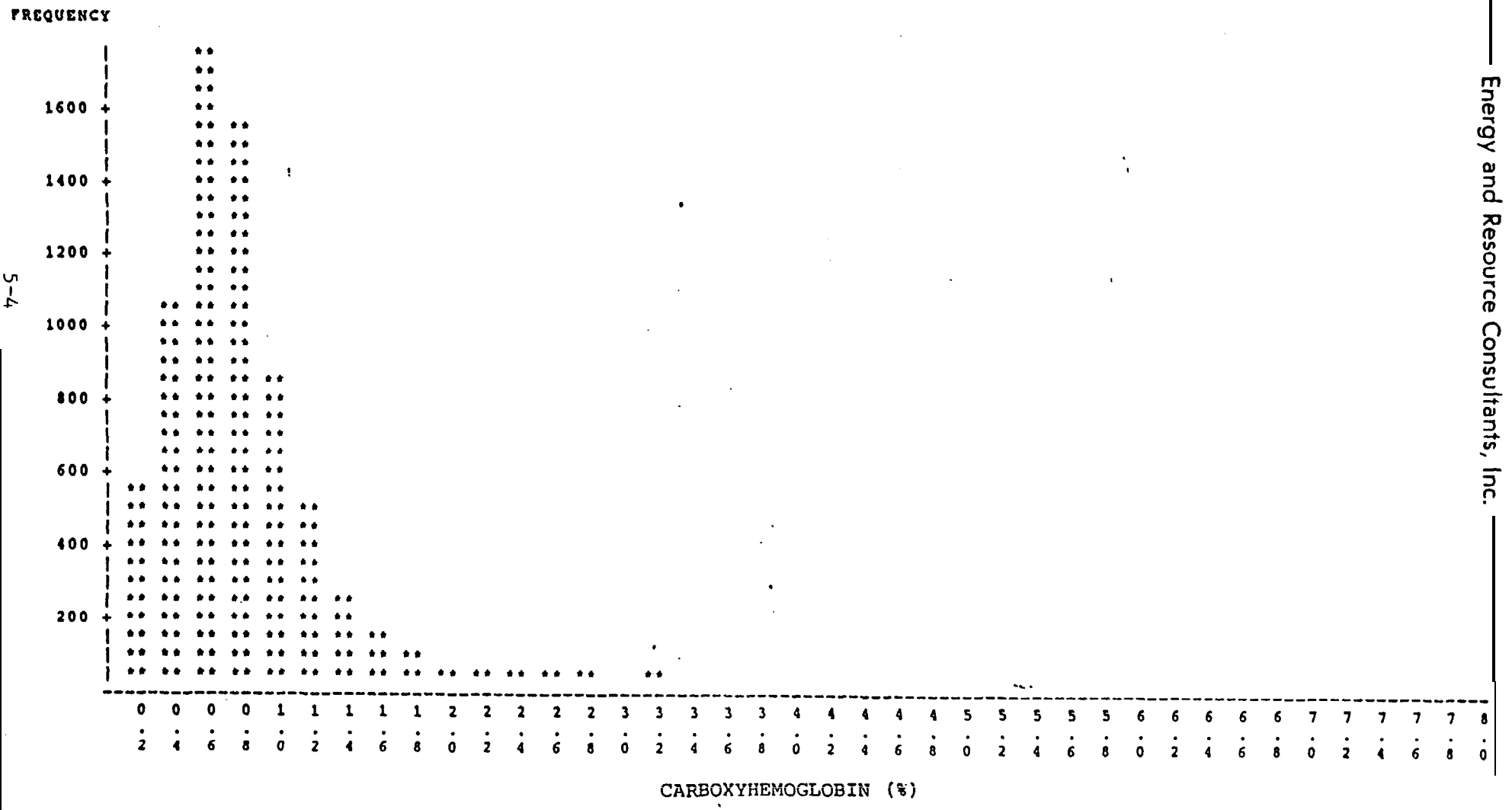


Figure 5-2

Frequency Distribution of COHb (%) For Smokers in NHANES II

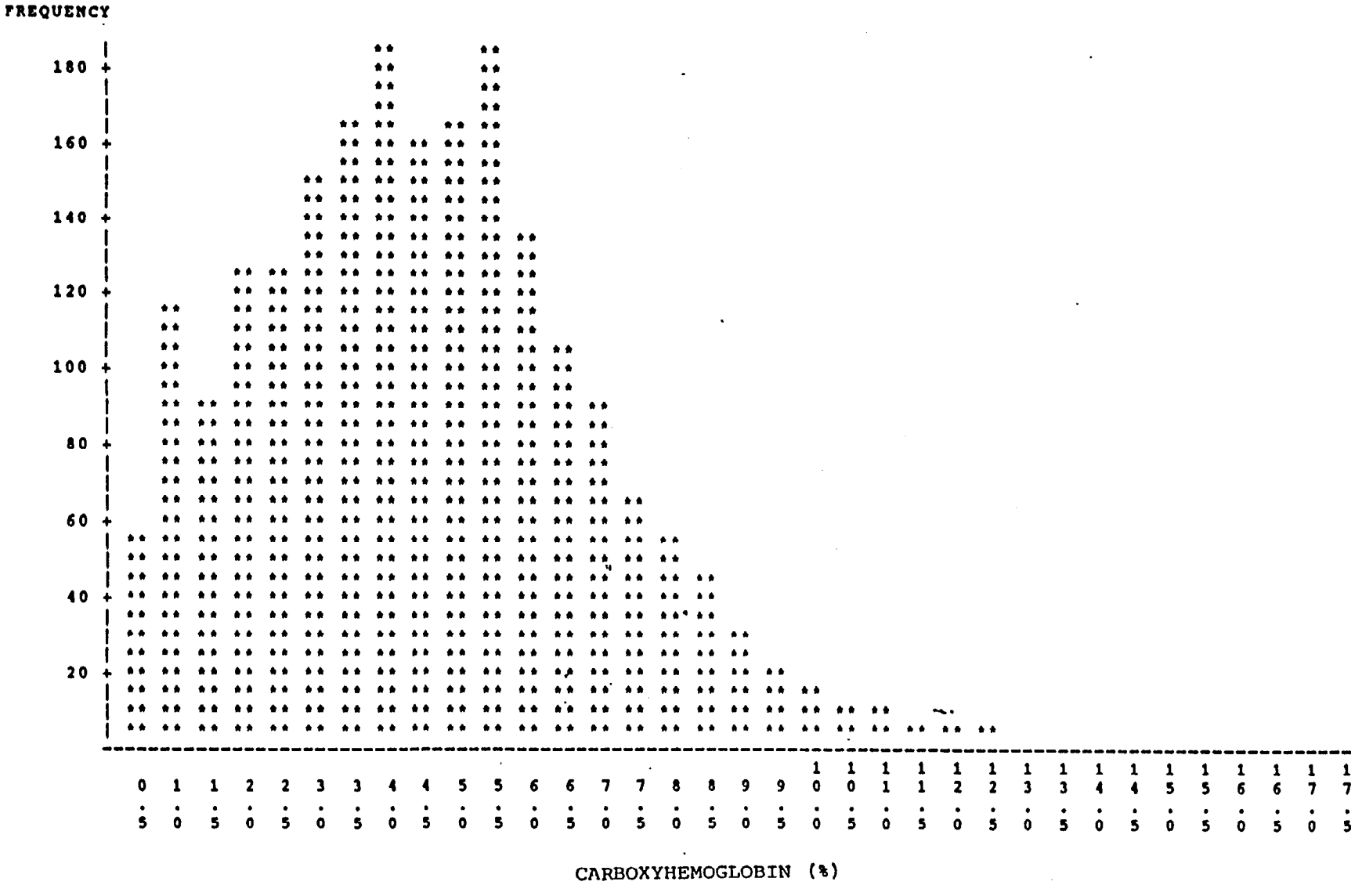


Table 5-1

COHb Levels from NHANES II

	Unweighted Geometric Mean	Unweighted Standard Deviation** (Geometric)	Weighted Geometric Mean***	N	Selected Values for this Analysis	
					Geometric Mean	Geometric Standard Deviation
All Locations* (1976-1980)						
Nonsmokers	.76	1.93	.78	4,875		
Smokers	3.78	1.96	3.80	1,479		
Santa Clara Co. (1976)						
Nonsmokers	1.01		1.01	110	1.01	1.93
Smokers	3.20		2.71	15	3.80	1.96

* All NHANES II with population > 100,000. Includes 44 U.S. Cities.
** The average of the geometric standard deviations calculated for each location.
*** Weighted with NHANES II weights to adjust for stratified sampling.

5.3 AMBIENT LEVELS OF CO IN SANTA CLARA COUNTY

Before discussing the health risks associated with the COHb levels presented in the previous section, it is important to note changes in ambient levels of CO that have occurred since these COHb measures were taken in 1976. It is also important to note the time of year these measures were taken due to the seasonal fluctuation in ambient CO levels.

Changes in CO from 1976 to 1985

Figure 5-3 shows that CO emissions and ambient CO levels have fallen significantly since 1976. The 1985 annual average of the daily maximum hourly CO reading is about half what it was in 1976 at both the San Jose and Gilroy monitors. The annual means of the daily high hour in 1976 were 7.4 ppm at San Jose and 3.7 ppm at Gilroy. In 1985 these were 4.2 and 1.7, respectively.

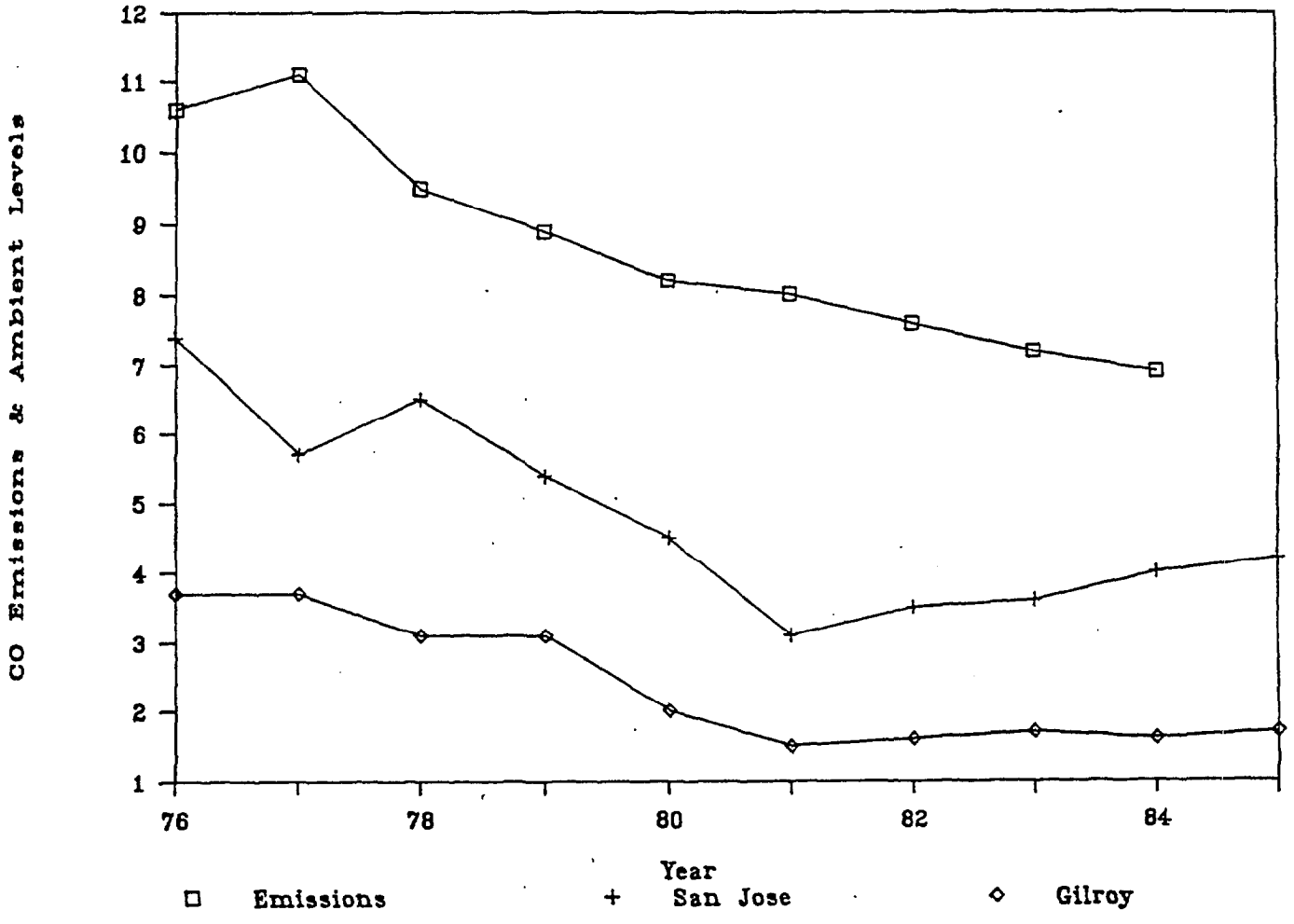
Available data on emissions and CO levels at stationary monitors in Santa Clara County indicate that the changes in these measures over time are highly correlated. The correlation between. county CO emissions and the CO measures for the San Jose monitor is .84 and the correlation between county CO emissions and the CO measures for the Gilroy monitor is .92, both correlations being significant at more than the 99% confidence level. Because the primary emissions source (automobiles) is distributed collinearly with population, and there is a high correlation over time between emissions and ambient conditions, it is reasonable to presume that a similar change in CO exposures from outdoor CO sources has also occurred. It should be noted that significant indoor sources of CO would remain.

As the data in Figure 5-3 indicate, CO levels are about twice as high at the San Jose monitor than at the Gilroy monitor. The San Jose monitor is located in the central city area, where the highest CO levels are likely to occur, but this monitor is probably more representative of ambient CO levels for most Santa Clara County residents than the Gilroy monitor. The vast majority of the Santa Clara County population (over 95%) resides in the northern half of the county, which is a fairly densely populated area that is more like downtown San Jose than like Gilroy in terms of traffic density. Thus, the San

Figure 5-3

Santa Clara County

CO Emissions and Ambient Levels 1976-85



NOTES:

1. Emissions are total CO emissions in Santa Clara County in 100 tons.
2. Ambient CO levels for San Jose and Gilroy are annual means of daily high hourly reading in ppm.

Jose monitor probably overstates ambient CO levels in the parts of the county where most people live, and the Gilroy monitor probably significantly understates the CO levels where most people live.

Seasonal Fluctuation in Ambient CO Levels

An important consideration in using the COHb levels measured by the NHANES II study is that there is a significant seasonal variation in ambient CO levels in Santa Clara County. The COHb measurements were made during a four-week period starting the last week in February. Table 5-2 shows the month-to-month variation in the average of the daily maximum hourly reading of CO, for 1984 and 1985. The pattern is fairly consistent. The highest CO levels are in January and December. Next highest are February, October, and November. Levels in March and September are somewhat lower still, and April through August have the lowest levels. If we give one-fourth weight to the February levels and three-fourths weight to the March levels, the CO levels during the time of the NHANES II examinations in Santa Clara County can be expected to be roughly half of the levels that occur during the peak months of January and December. This means that the COHb levels measured in late February and March may be reasonably representative of levels that occur under average CO conditions, but will be likely to significantly understate the levels that, might occur during the peak CO season.

Combining the change in CO levels from 1976 to 1985 and the seasonal fluctuation in CO levels, suggests that the CO levels during the peak CO months in 1985 are likely to be in the same range as the levels during the NHANES II examination in February and March 1976. This would also suggest that the range of COHb levels across the sample population in February/March 1976 might be representative of expected levels during the peak CO months in 1985. This is the assumption made to facilitate the subsequent preliminary analysis.

Measured CO Levels in 1985 Versus the Standards

In 1985, the Federal standards for the 8-hour average CO level were exceeded on 16 days. The California standard, which is slightly more stringent than

Table 5-2

Seasonal Fluctuation in Ambient CO

	Monthly Mean of Daily Max Hour (ppm)											
	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
San Jose												
1984	7.9	5.5	3.9	2.4	2.1	1.7	1.9	2.1	5.0	4.2	4.9	6.9
1985	8.6	5.2	2.8	2.4	1.7	1.8	2.0	2.1	2.7	4.4	5.6	11.0
Gilroy												
1984	3.1	1.8	1.4	1.0	1.0	1.0	0.9	1.1	1.7	1.5	2.0	2.7
1985	3.5	2.4	1.3	1.0	1.0	0.8	0.9	0.9	1.0	2.0	2.3	3.5

LOWEST CO SEASON

PEAK CO SEASON

the Federal standard, was also exceeded. The Federal standard is met if the second high 8-hour average for the year does not exceed 9.2 ppm. The second high for 1985 at San Jose was 14.3, so a 36% reduction in this measure would have met the Federal standard. The California standard is met if the highest 8-hour average for the year does not exceed 9.0 ppm. The highest 8-hour average for 1985 at San Jose was 15.6, so a 42% reduction in this measure would have met the California standard.

In 1984, Gilroy had an 8-hour high of 3.4 ppm. This is the lowest annual high 8-hour level recorded in Santa Clara County during 1981-1985. Compared to the 8-hour high at the San Jose monitor in 1985, this would mean a 78% reduction if the 3.4 level were to occur in San Jose.

5.4 ESTIMATES OF THE POPULATION ABOVE SELECTED COHb LEVELS

In this section we present estimates of the number of people at risk of adverse health effects due to COHb levels measured in 1976. This is not the number of people who would be expected to experience such health effects, but is the number of people who are at some increased risk relative to others with lower COHb levels. This is different than the estimates presented for other pollutants in this report, which are estimates of the number of pollutant related health incidents in a year. Not every person at risk would experience a CO related health incident, but some individuals might experience more than one incident in a given time period. Since the NHANES II data were collected over a four week period, the estimates in this section are appropriately interpreted as the number of people at some increased risk of one or more health incidents during a four week period. As discussed below, these COHb levels are likely to be indicative of levels occurring during the peak CO season in 1985.

Extensive reviews of the literature concerning the health effects of CO have been prepared by the U.S. Environmental Protection Agency (U.S. EPA, 1979, 1983) and the California Air Resources Board (CARB, 1982). The discussion in this section relies upon the conclusions presented in these reviews.

Information concerning the lowest COHb levels at which certain effects have been observed is available for three types of effects: (1) reduced time to onset of angina pain for heart disease patients, (2) reduced exercise capacity, and (3) reduced vigilance. Effects on fetuses have been observed in studies of smoking mothers, but the effects of CO alone have not been isolated. Individuals with chronic lung diseases and chronic anemia are also suspected of being at greater risk than the general population, but quantitative evidence is insufficient to use in this analysis.

Several studies have found that the time to onset of angina is reduced in some subjects with heart disease when they are exposed to controlled amounts of CO. This has been reported at COHb levels as low as 2-3%, but questions have been raised about the validity of several of the studies that have found these effects, especially below 2.5%. For this analysis we have selected a range of 2.5% to 3.0% as the COHb level above which individuals with heart disease could be expected to be at increased risk.

The significance of the reduced time to onset of angina pain is not entirely clear, but it suggests that oxygen supply to the heart is reduced enough to cause some stress. For this analysis, we presume that all heart disease patients would be at increased risk of ill effects even if they do not typically experience angina pain. The California Hypertension Survey of 1983 by the California Department of Health Services indicates that 10% of the California population 18 years or older has ischemic or cardiovascular heart disease. For Santa Clara County in 1985 this implies about 100,700 individuals.

Several studies have found that exercising capacity in healthy individuals is reduced at elevated COHb levels. Shorter time until maximum aerobic capacity is decreased has been observed at as low as 4% COHb. All individuals who exercise would be subject to this risk at COHb levels of 4% or more. We use an estimate of 40% of the adult population who regularly exercise, but at any one point in time only a very small fraction of these individuals would be engaged in vigorous exercise.

There is some evidence that a healthy individual's capacity to carry out complex tasks is also impaired at elevated COHb levels. This has been observed at COHb levels as low as 5%. At levels this low the effect is not strong, but could be enough to affect complex tasks such as driving.

The estimated percent of the population at or above each of the selected COHb levels based on the 1976 NHANES data is shown in Table 5-3. These estimates assume a lognormal distribution on the 1976 COHb geometric means and geometric standard deviations, which were presented in Table 5-1. The percentage estimates are the percent of nonsmokers and smokers expected to be above the selected COHb levels based on the geometric means and standard deviations presented in Table 5-1.

Estimates of the number of people at risk of experiencing each of the three effects during a four-week period were obtained by applying these percentage estimates to the relevant population estimates for Santa Clara County in 1985. An estimate that 30.5% of the adult population smokes was based on state-wide data from the California Department of Health Services. It was assumed that all of the estimated 10% of the adult population with heart disease would be in the nonsmoker group, since everyone with diagnosed heart disease is advised to quit smoking. It was assumed that the estimated 40% of adults who exercise regularly would be evenly distributed between smokers and nonsmokers, an assumption that probably results in an understatement of the number of nonsmokers potentially affected (and overstatement of the number of smokers potentially affected), since nonsmokers are more likely than smokers to be exercising regularly.

Although there is some information available about how an individual's COHb levels change when exposed to controlled changes in ambient CO (Coburn et al., 1965), this information is not sufficient to predict with any confidence how the COHb levels for the population would be expected to change if CO emissions were reduced enough to meet the Federal or California ambient CO standards. We expect that average COHb levels and the number of people experiencing COHb levels high enough to have increased risk of health effects would be lower if ambient CO levels are lower, but how much lower is uncertain for the following reasons.

Table 5-3
Estimated Number of Adults at Risk of Potential Health Effects Associated with Elevated COHb

	Nonsmokers	Smokers
Number of Adults in Santa Clara County*	699,800	307,100
Angina Effects		
% of Individuals with COHb > 2.5-3%	5-8.5%	63-72%
Relevant Population = Heart Disease Patients (10% of all adults)	100,700	--
Number of Adults at Risk of Potential Angina Effects	5000-8,600	--
Exercise Effects		
% of Individuals with COHb > 4%	2%	46%
Relevant Population = Adults Who Exercise (40% of all adults)	279,900	122,800
Number of Adults at Risk of Potential Exercise Effects	5,600	56,500
Vigilance Effects		
% of Individuals with COHb > 5%	0.8%	34%
Relevant Population = All Adults	699,800	307,100
Number of Adults at Risk of Potential Vigilance Effects	5,600	104,400

* All numbers are rounded to the nearest hundred.

- People spend only a fraction of the day outdoors. Because there are significant indoor sources of CO that are independent of ambient levels outdoors, there is uncertainty about how changes in outdoor CO levels would affect the individual's overall COHb levels, which are expected to be a function of both indoor and outdoor exposures.

- CO levels, and therefore COHb levels, fluctuate significantly hour to hour and place to place. There is not sufficient information available to estimate how the range of COHb levels in the population would be expected to change as CO levels at the stationary monitors change. The standard deviation as well as the mean level of COHb is needed to predict the number of people expected to experience COHb above selected levels at which risks are expected to increase.

- Because it takes time for COHb levels to change in response to changes in ambient CO levels, it is uncertain what averaging time (e.g., 1-hour or 8-hour) is most appropriate for measuring CO exposure.

We do not present quantitative adjustments in these estimates to account for the changes in CO exposure that have occurred from 1976 to 1985, or that would have been expected if the standards had been met, but the information presented in the previous section gives an idea of the magnitude of the changes in ambient CO levels involved. The change in ambient CO from 1976 to 1985 and the observed seasonal fluctuation suggests the estimates based on the 1976 NHANES II data are indicative of the number of people likely to be at risk of potential health effects during a four-week period in the peak CO season during 1985. The percentage reductions in 1985 ambient CO levels required to meet the Federal and California standards are 36% and 42%, respectively. The number of people above the selected COHb levels would fall by more or less than this amount depending on how the distribution of COHb levels would change. If the standard deviation of COHb fell, as well as the mean COHb, then it is conceivable that meeting the standards might reduce the risks to nonsmokers from outdoor sources of CO to near zero.

One thing that stands out in the estimates presented in Table 5-3 is the dramatic difference between the number of nonsmokers at risk and the number of smokers at risk. It is clear that smoking has a far greater effect on COHb levels than does ambient CO levels, and the number of smokers with elevated COHb levels would still be significant even if ambient CO levels were reduced to zero.

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6.0 DOLLAR VALUES FOR CHANGES IN VISIBILITY AESTHETICS

6.1 INTRODUCTION

The aesthetic effects of air pollution are an important component of the overall impact of manmade air pollution in the atmosphere. Small particles and gases in the atmosphere can form plumes, layered haze, and regional haze, which can cause changes in visual range, contrast, light extinction and color. Visibility is not something that can be directly bought or sold, but evidence suggests that people value good visibility. In urban and residential areas, a nice view can add considerable value to a property. In recreation areas people drive and hike considerable distances in order to reach certain overlooks and view the scenery.

Research results are available that give estimates of what individuals are willing to pay to have improvements, or prevent deterioration, in visibility in areas where they live and in national parks and recreation areas that they visit. Available evidence is used in this chapter to estimate the value to the public of improvements in visibility in Santa Clara County that would be expected to occur in association with the potential decreases in particulate matter presented in Chapter 2.

In estimating benefits of improved visibility in California, Rowe et al. (1986) found that the estimates for visibility in residential areas significantly outweighed the estimates for visibility related to recreation in national and state parks and forests. We have therefore estimated values for residential areas only for Santa Clara County. These estimates can be expected to include values related to activities undertaken in local parks near people's homes.

6.2 PREDICTED CHANGES IN VISIBILITY FOR THE ALTERNATIVE PM SCENARIOS

There is considerable complexity involved in estimating the benefits of good visibility. From the aesthetic point of view, people are as concerned with what they can see through the air as with what is in the air. Emissions of

pollutants must therefore be translated into effects that are visually perceivable before they can be evaluated. The content of the view and the interests of the individual viewing it can affect the perception of visibility conditions, as can many other circumstances such as weather and time of day. For this analysis, predicted changes in PM were used to estimate changes in visual range that would be expected to occur for each scenario. Visual range is one aspect of perceptible visibility, but it does not reflect everything the viewer might perceive, such as color and contrast. Visual range is the most useful way to characterize visibility for this analysis because most of the visibility values research done to date has defined and valued visibility in this way.

Trijonis (1982) has estimated a relationship between visual range and particulates and humidity based on data for California. The relationship is as follows:

$$\left[VR = 24.3 / \left(.12 + .04 * \frac{SO4}{1 - \frac{RH}{100}} + .03 * \frac{NOX}{1 - \frac{RH}{100}} + .003 * \frac{T}{1 - \frac{RH}{100}} \right) \right] \qquad (6\text{-}1)$$

where:

VR = visual range in miles
SO4 = sulfates in $\mu g/m^3$
NOX = nitrates in $\mu g/m^3$
T = TSP - SO4 - NOX in $\mu g/m^3$
RH = relative humidity in %

We have simplified Equation (6-1) by substituting data for Santa Clara County. Data were available for sulfate and nitrate levels as well as TSP levels for 61 days in 1985 for the San Jose and Moorpark monitors. The levels of sulfates and nitrates were highly correlated with the levels of TSP. R-square statistics in simple regressions relating sulfates as a function of TSP were .89 and .86, and relating nitrates as a function of TSP were .82 and .88. Average ratios of

sulfates to TSP and nitrates to TSP were calculated to allow Equation (6-1) to be written in terms of TSP. The following averages were obtained.

	Average Daily Ratios	
	Sulfates/TSP	Nitrates/TSP
San Jose	.07	.09
Moorpark	.09	.11

Published data from the National Oceanic and Atmospheric Administration on humidity levels in selected California cities for 1980 indicated average levels of 71-73% in San Francisco. Other California cities (San Jose not included) reported average annual levels of 65-75%. The value selected for this analysis was 70% for Santa Clara County.

Using the following values, Equation (6-1) can be simplified to Equation (6-2).

SO4 = .08 * TSP
NOX = .10 * TSP
T = .82 * TSP
RH = 70

VR = 24.3/(.12 + .0289 * TSP) (6-2)

Equation (6-2) was used to estimate levels of visual range for each of the PM scenarios. The annual arithmetic means from Table 2-1 were used.. The predicted visual range estimates for each of the monitors are shown in Table 6-1. Actual visual range data from the San Jose airport indicate an annual average of about 6.3 miles, while the predicted visual range using Equation (6-2) for San Jose is 7.9 miles. This suggests that Equation (6-2) may be slightly overstating actual visual range levels, but given that Equation (6-2) is being used to evaluate changes in visibility across scenarios, the error is probably not large. It is also important to note that using Equation (6-2) to predict changes in visual range associated with changes in TSP assumes that when TSP changes, sulfates and nitrates will change in proportion.

Table 6-1
Predicted Visual Range Levels (Miles)
for Santa Clara County

	4th Street Monitor	Moorpark Monitor
1985 Actual PM Baseline	7.9	13.3
1985 Adjusted PM Baseline	9.9	13.3
Meeting Federal Standard	9.9	13.3
Meeting California PM Standard	16.1	16.1
Attaining Low PM Levels	19.5	19.5

6.3 VISIBILITY VALUE FUNCTION

Two types of studies have been used to estimates willingness-to-pay values for changes in visibility, or air quality in general. One type is the contingent valuation approach in which subjects are asked to estimate directly how much they would be willing to pay for a specified change in visibility, usually described to them and illustrated with photographs. The second type is the property value study in which prices of residential properties are analyzed to determine how they vary across locations with different levels of air quality, everything else being equal.

Both of these estimation approaches are reviewed in detail in Rowe and Chestnut (1982). Each has important limitations, but repeated applications have been made and some consistency is being found in the results. For this analysis we rely primarily on the results of contingent valuation studies concerning the value of visibility in urban residential areas. These studies are reviewed in Rowe et al. (1986). In each of these studies subjects are interviewed and shown photographs of alternative levels of visibility in the areas in which they live. They are then asked what they would be willing to pay, usually each month, for their households to have a specified improved level or to avoid a specified deterioration. An important concern in these studies is whether subjects are able to separate concerns about visual aesthetics from concerns about the health effects, and other unpleasant things like odor, that may also be associated with different levels of air pollution. To address this concern, some studies have asked subjects to give values for all reasons and then asked them to divide this amount among health and visual aesthetics. In other studies the subjects have been specifically asked to think only about visual aesthetics, not health effects.

Table 6-2 lists the contingent valuation studies that have been conducted in the U.S. and gives the cities in which each was conducted. Based on the results for the studies shown in Table 6-2, Chestnut et al. (1986) estimated the value function shown in Table 6-3 for changes in visibility levels.

Table 6-2
Contingent Valuation Studies of Visibility Values
in Residential Areas

Study	City
Brookshire et al. (1979)	Los Angeles
Loehman et al. (1981)	San Francisco
Randall et al. (1981)	Chicago
Rae et al. (1983)	Cincinnati
Tolley et al. (1984)	Atlanta Boston Miami Cincinnati Mobile Washington, D.C.

Table 6-3

Summary of Results of Contingent Valuation Studies

Concerning Visibility in Residential Areas

Variable	Coefficient
ln (v2/v1)	195.10* (25.11)
RANK	253.59* (72.45)
DIST	-94.96 (70.87)
PRETEST	21.88 (52.10)
WEST	-5.33 (48.45)
N = 36 R^2 = .78 F = 21.42	

* significant at 99%.

Variable Definitions

Dependent Variable = Annual willingness to pay per household in 1984 dollars.
V1 = the initial level of visual range in miles
V2 = the new level of visual range in miles
RANK = contingent ranking method was used in study: 1=yes; 0=no
DIST = visual range was presented as a distribution of several levels, not a single average level: 1=yes; 0=no
PRETEST = study was a pretest for a larger effort: 1=yes; 0=no
WEST = study was conducted in the western U.S.: 1=yes; 0=no

The equation shown in Table 6-3 implies that the value to the household is a function of the percentage change in visibility, and is constant for a given percentage change. The percentage change in visual range may be a good way to characterize a person's perception because it takes into account the starting point and the size of the change. Other functional forms were also found to fit the results of the contingent valuation studies, but they would predict similar values. The four variables in addition to the visual range variable represent different characteristics of the studies. Only RANK was found to be statistically significant. This reflects the results of an estimation technique that was used in only one of these studies and may have had a significant upward bias on the results. For the calculations in this analysis, RANK and the insignificant variables were set equal to zero.

For this analysis, the coefficient on ln(V2/V1) was adjusted to 1985 dollars. The upper and lower estimates were obtained by calculating the 95% confidence interval for this estimated coefficient. This interval is plus or minus 1.96 * 25.1. The lower, best and upper estimates for this analysis were calculated as follows (in 1985 dollars). The 1985 baseline visual range levels given in Table 6-1 are used for V1, and V2 values are the visual range estimates for each alternative pollution level.

$$\text{Lower Annual Value per Household} = 151 * \ln (V2/V1) \quad (6\text{-}3)$$

$$\text{Best Annual Value per Household} = 202 * \ln (V2/V1) \quad (6\text{-}4)$$

$$\text{Upper Annual Value per Household} = 253 * \ln (V2/V1) \quad (6\text{-}5)$$

There are three property value studies that have been conducted in California concerning the effects of air quality on residential property values (Brookshire et al., 1979; Loehman et al., 1981; and Trijonis et al., 1984). These studies have found a significant relationship between residential property values in San Francisco and Los Angeles and different measures of air quality. It is expected that the values obtained in these studies will reflect both health and visual aesthetics, but the results provide important support for the credibility of the magnitudes of the responses obtained in the contingent valuation studies. Even if some adjustment is made to split the values between health and visual

aesthetics, the results of the property value studies are in the middle to upper range of the contingent valuation results for comparable changes in visual range.

6.4 RESULTS

The estimates of total dollar value for the changes in visibility expected to be associated with attaining the alternative reductions in PM in Santa Clara County are shown in Table 6-4. Estimates are given for both the actual 1985 baseline TSP and the adjusted 1985 baseline TSP (see Chapter 2 for an explanation of the adjusted baseline). For the whole county, the best estimate for meeting the California PM standard using the actual baseline implies a value of about $43 per household. Since 56% of this value occurs in San Jose, and the population is about 50% in San Jose, estimated values for households in San Jose are somewhat higher than for those in the remainder of the county. This is due to larger predicted visual range changes in San Jose than in the rest of the county under the alternative scenarios.

Table 6-4
Household Values for Changes in Visibility
(Thousands of Dollars)

	Actual 1985 Baseline		
	Meeting Federal Standards	**Meeting California Standards**	**Meeting Lowest Levels**
San Jose			
Lower	785.0	9142.2	16493.2
Best	1050.2	12230.0	22063.7
Upper	1315.3	15317.8	27634.2
Remainder Of County			
Lower	8.9	7308.9	14617.9
Best	11.8	9777.4	19555.1
Upper	14.8	12246.0	24492.3
Total County			
Lower	793.9	16451.1	31111.1
Best	1062.0	22007.4	41618.8
Upper	1330.1	27563.7	52126.5

	Adjusted 1985 Baseline		
	Meeting Federal Standards	**Meeting California Standards**	**Meeting Lowest Levels**
San Jose			
Lower	0.0	8357.2	15708.1
Best	0.0	11179.8	21013.5
Upper	0.0	14002.5	26318.9
Remainder Of County			
Lower	0.0	7300.0	14609.1
Best	0.0	'9765.6	19543.3
Upper	0.0	12231.2	24477.4
Total County			
Lower	0.0	15657.2	30317.2
Best	0.0	20945.4	40556.8
Upper	0.0	26233.6	50796.4

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7.0 MATERIALS DAMAGE ASSOCIATED WITH OZONE AND TOTAL SUSPENDED PARTICULATE MATTER[1]

7.1 INTRODUCTION

The effects of air pollution on man-made materials vary depending upon the composition of the material, the pollutant and the characteristics of exposure. Ozone, TSP, sulfur compounds and NO_2 have all been identified with materials damage, however only ozone and TSP are considered in this analysis because NO_2 and SO_2 levels are well below the standards in Santa Clara County.

Reduction in social welfare due to air pollution induced materials damage is usually expressed in terms of one or both of the following general classes of costs to producers and consumers:

- accelerated replacement, and maintenance and repair costs, due to reduced service life or degradation of the appearance of a material. This also includes cleaning of soiled materials.

- increased avoidance costs when industries use additives, preservatives and reformulations to avoid or minimize damage.

The principal damages associated with TSP include corrosion and soiling of metallic and non-metallic building materials; and the soiling, fading and discoloration of fabrics and works of art. The analysis here uses upper and lower bound estimates of soiling damages incurred by households based upon work by Manuel et al. (1982). This study estimated a system of demand equations for the consumption and production of cleanliness that directly address household adjustments to changes in air quality. Other substantiating evidence is identified.

Materials that can be adversely affected by ozone include elastomers, paints, fabrics, dyes, artists' pigments, polyethylene and various types of plastics.

1. For more detailed discussions, the reader is referred to Chapter 7 in Rowe al. (1986) written by D. Peterson, which discusses in depth the measurement of physical and economic damages, and the selection of damage functions. The review here is an abbreviated version of that presentation.

The economically most significant class of materials affected by ozone appears to be elastomers, and more specifically automobile and truck tires (EPA, 1983; Rowe et al., 1986). In the following analysis, ozone damage is estimated primarily using damage functions relating annual average concentrations and tire damage.

7.2 TSP INDUCED MATERIALS DAMAGE

Materials at Risk to TSP

TSP has been identified as causing soiling and discoloration effects on a wide variety of materials, including paint, structural metals, fabrics and other building materials. The primary effect is considered to be the soiling of surfaces. This soiling has welfare impacts through increased cleaning costs and by reducing the useful life of affected materials.

Very few physical dose-response or damage functions have been developed for materials soiling. Among the few such studies is work by Beloin and Haynie (1975) examining soiling damage to painted cedar siding, concrete block, brick, limestone, asphalt shingles and window glass. Unfortunately, this work did not account for variations in humidity across sites and has seldom been used in damage assessments.

TSP Damage Functions

Economic analyses of TSP induced materials damage can be classified as either cleaning frequency and expenditure models, or economic supply and demand models. The cleaning frequency models determine the reduction in household cleaning and maintenance costs affected by a decrease in particulate concentrations assuming a fixed level of cleanliness is desired. The supply and demand models assume cleaning related materials and labor purchased are determined by the demand for cleanliness, the prices of cleaning materials and labor, as well as the prices and demand for all other competing goods and services in the household's operating budget.

Cleaning costs alone can be expected to understate the benefits of reduced soiling because they do not incorporate aesthetic and psychological benefits associated with changes in soiling levels and cleaning activities.

Upper and lower bound estimates. The upper and lower bound estimates are taken from a study by Manuel et al. (1982), which bounds the estimates of other previous studies. In this work a system of demand equations for household consumption of goods and services, including cleanliness, is estimated with data from the 1972-73 U.S. Survey of Consumer Expenditures and on information on price and quantity indices for cleaning and other household goods and services. The model includes sectors covering food purchases, shelter maintenance, home operations, home furnishings and equipment, clothing purchase and dry cleaning, transportation, and health and personal care. The home operations sector has expenditure equations covering laundry and cleaning products and other household products. The cleaning equations relate the expenditures on laundry cleaning and other household products to their prices, TSP measures, household variables and price indices for other sectors. The focus of the analysis is upon the expenditure and activity tradeoffs households make when air pollution levels change, from which.benefit measures of the air pollution change are derived. This work is selected as the key study in this damage assessment as it generates benefit measures more consistent with the desired theoretical measures than are available through other approaches and because it has formed the basis of EPA materials benefit assessment for TSP in regulatory analysis.

Based upon the Manuel et al. work, the following upper and lower bound estimates are selected.

TSP Materials Damage Lower Bound Function

$$(\text{Change in } Y) = 14.80 * (\text{Change in TSP2}) \qquad (7\text{-}1)$$

where:

Y = annual household damages ($1985)
TSP2 = annual second high 24 hour reading in $\mu g/m^3$

TSP Materials Damage Lower Bound Function

(Change in Y) = .52 * (Change in TSP2) (7-2)

where the variables are defined as above.

Best estimates. The best estimate is taken as the average of the upper and lower bound estimate based upon Manuel et al. (1982). This middle range is also roughly consistent with other earlier work on TSP materials damage briefly reviewed below. Note, however, that the Manuel et al. work measures damages based upon changes in the annual second high 24-hour TSP reading, while the studies below use changes in the annual average 24-hour reading. Therefore, the damage estimates are not directly comparable.

In one of the earliest TSP damage function studies, Michelson and Tourin (1976) compared frequencies of household cleaning and maintenance activities in Steubenville, Ohio and Unionville, Pennsylvania. Questionnaires were sent to residents of both areas covering demographic data and cleaning frequencies. Based upon the differences in ambient TSP levels and cleaning costs, a per capita damage estimate of $2.12 ($1985) per $\mu g/m^3$ change in annual average TSP concentration was calculated. This study has been subsequently criticized for potential biases and omissions by Wadell (1974), Freeman (1982) and others. Moreover, the pollution levels compared in the study are well above those in the Santa Clara County study region and may not be readily extrapolated to the current study.

Booz, Allen and Hamilton (1970) developed an extensive soiling data base with 1090 households in the Philadelphia SMSA that has been reanalyzed in several studies. The survey area covered four different pollution zones and different sociodemographic groups. Booz, Allen and Hamilton (BAH) used this data to analyze the relationships between cleaning frequency and pollution levels and concluded that the alternative pollution levels in the study region, ranging from < 75 $\mu g/m^3$ to > 125 $\mu g/m^3$, had no measurable effect on out-of-pocket cleaning and maintenance expenditures.

The BAH conclusions have been found unmerited based upon methodological and empirical reanalysis of the work (Liu and Yu 1976, Ryan et al. 1981, Cummings et

al. 1981, and Watson and Jaksch 1982). The BAH analysis focused upon activities not necessarily sensitive to changing TSP levels while ignoring potentially sensitive activities, such as vacuuming, thus obscuring the underlying relationship of interest. The analysis ignored household cost of labor and ignored disutilities associated with soiling and cleaning. Further, other important control variables were also ignored.

Cummings et al. (1981) reanalyzed 11 of the cleaning tasks surveyed in the BAH survey that were most likely to be associated with the types of soiling experienced due to air pollution. Cummings also considered both out of pocket expenditures and labor time associated with cleaning. Data were collected in each of the four BAH pollution zones on the number of households performing the different tasks and the frequency of task performance. For each task, Cummings et al. reported the number of households where the task was performed by the household and the number where the task was performed by outside cleaning help. Total costs were determined as the sum of the costs incurred by both types of households. The labor cost in do-it-yourself households and the time spent performing the tasks were developed in a new survey with 30 respondents from each of the four BAH pollution zones.

By relating ambient pollution concentrations in each of the four BAH pollution zones to imputed cleaning costs of out-of-pocket expenditures and time, Cummings et al. developed a linear economic.damage function. This function suggests marginal damages of $8.20 (1985) per $\mu g/m^3$ change in annual average TSP per household per year. Cummings et al., however, note that this relationship is simplistic, covers only selected damages and must only be viewed as indicative of potential damages.

Significant biases and omissions. The damage estimates focus upon cleaning efforts and expenditures by households. As such, values associated with aesthetic and psychologic impacts of soiling or increasing cleaning activities may be omitted. Also omitted are cleaning damages incurred at commercial and industrial buildings. As a result, the estimates presented here are likely to be biased downward.

7.3 OZONE INDUCED MATERIALS DAMAGE

Materials at Risk to Ozone

Elastomers. A number of commercially important rubber-based products are fabricated from unsaturated elastomers with well-known sensitivities to ozone damage. These include tires, rubber seals and pharmaceutical goods. Vehicle tires have been identified as the single major user of sensitive elastomers with potential significant economic costs associated with ozone (McCarthy et al., 1983).

Oxidation affects tires most significantly by causing cracks to develop in tire sidewalls. Flexible unsaturated rubbers are placed over the strength-bearing cores in a tire's sidewall. However, such unsaturated rubber can be susceptible to ozone damage. To minimize such damage, tire manufacturers add anti-oxidant materials to such rubber formulations. Benefits from reduced concentrations of ozone can be expected by tire manufacturers and tire retreading firms. The manufacturers would benefit by decreasing the quantity of anti-oxidant required to protect tires through their predicted use lifetimes; and tire retreaders would benefit through increased numbers of usable tire carcasses (Rowe et al., 1986).

Other materials. Damage to other materials is often associated with ozone; but total damages in these categories are believed to be economically less significant than for elastomers, or are unknown.

- Ozone is known to adversely affect the strength of cotton, nylon and acrylic fibers and to cause certain sensitive dyes to fade. While the economic value of fabric and dye fading impacts is uncertain, the damages are believed to be relatively small (Rowe et al., 1986).

- Several pigments used in artwork have been found to fade when exposed to ozone concentrations typical of outdoor ambient conditions and, it is asserted, will likely be sensitive to typical indoor levels in unprotected buildings over several years in areas with high outdoor concentrations (Shaver et al. 1983, Drisko et al. 1984). Because art work often requires protection for hundreds of years, ozone

concentrations in unprotected buildings may pose a significant risk of damage to art collections.

- Studies indicate that vinyl and acrylic oil coatings (paints) may be affected by ozone, but the effect to both the coating and the the material coated is minimal (McCarthy et al. 1981, Haynie and Spence 1984).

- Other potentially sensitive materials include asphalt, dried milk, recording tape, and polyethylene (NAS 1977), but insufficient evidence exist to quantify the degree and importance of these risks.

Ozone Damage Functions

Lower Bound Estimates. The lower bound damage estimates are calculated based upon the work of McCarthy et al. (1983) for tire damage. The McCarthy analysis calculated the benefits based upon assumptions on the required level of anti-oxidants in tires at alternative ozone levels, the price of anti-oxidants per pound, tire use-life and annual tire production based upon national statistics and regional statistics for the East. Retread related damages were calculated based upon data on tire sales, discards sales, and prices of tire casings. In Rowe et al. (1986), the McCarthy analysis was updated with available southern California data on tire sales and annual per capita damage functions generated. This damage function, in 1985 dollars, is reported in Equation 7-3 for a change in ozone.

Ozone Materials Damage Lower Bound Function

$$(\text{Change in } Y) = 8.68 * (\text{Change in ozone}) \qquad (7\text{-}3)$$

where:

Y = annual per capita damages ($1985)
ozone = annual average ozone all hours measured in ppm.

Among the limitations in this function are that evidence suggests other elastomers and other materials may be affected, and that the elastomer damage

function may be increase exponentially, rather than linearly, at higher ozone levels.

Upper Bound. Studies by Mueller and Stickney (1970), NAS (1977) and Stankunas et al. (1982) have estimated the economic value of elastomer damage due to ozone substantially larger than in McCarthy et al. In addition, studies by Barrett and Waddell (1973), Salmon (1970), Waddell (1974) and others have suggested that damages to fabrics, dyes and other materials, when considered, would substantially increase estimated damages by a factor of 6-10 over those estimated by McCarthy et al. However, some of these estimates may have been little more than guesswork based upon limited scientific evidence.

Based upon the work of Stankunas et al., a per capita annualized damage function is selected to represent the lower end of the upper bound estimates.

Ozone Materials Damage Upper Bound Function

(Change in Y) = 47.43 * (Change in ozone) (7-4)

where:

Y = annual per capita damages ($1985)
ozone = annual average ozone for all hours in ppm

Best Estimate. The best estimate is selected as the midpoint of the upper and lower bound estimates. This is made with the consideration that the lower bound estimate may be the most defensible estimate for those elastomer damages included, but that potential damages to other elastomers, artwork, fabrics and dyes are excluded in the lower bound estimate.

Significant Biases and Omissions

The lower bound estimates may be biased downward by omitted damage categories, while the upper bound estimates may be significantly biased upward due to use of damage function estimates with limited scientific foundation.

7.4 RESULTS OF THE MATERIALS DAMAGE ASSESSMENT

The estimates of predicted reductions in materials damage for alternative reductions in particulate matter and ozone are shown in Table 7-1. The alternative levels of TSP are the same as for the health calculations discussed in Chapter 2. Estimates for TSP are given for the actual 1985 baseline and the adjusted 1985 baseline (see Chapter 2 for an explanation of these two baselines). The predicted change in the second high 24-hour TSP reading for each scenario is shown in Table 2-1.

The estimates for ozone damages are based on the percentage reductions that would bring the daily high hourly levels into compliance with the hourly standards. Changes in the annual average of all hours are calculated presuming that all hours above .04 ppm would be reduced by the same percentage change that would bring the high hour into compliance, but no hour was reduced below .04 ppm. Because ozone data are not collected at three of the five monitors in the county during December, January, and February, an adjustment was made to obtain annual averages for all five monitors. Data from the Alum Rock station (with generally fewer exceedences of standards than Los Gatos) were substituted for the missing months of data for the other three stations. Although this is an arbitrary procedure, we judged it preferable to dropping the data from the three monitors that are not complete. The error is expected to be small since ozone is very low in the three months for which data were missing. The estimates of the annual average ozone for each scenarios are shown in Table 7-2.

The total best estimate for meeting the California standards is about $75 million based on the actual baseline, and about $67 million based on the adjusted baseline. This amounts to about $48 to $54 per person on an average in the county, but about 68% of the total occurs in San Jose. The difference between the TSP and ozone benefits is dramatic, with the ozone amounting to about $.05 per person.

Table 7-1
Dollar Value Estimates for Predicted Changes
in Material Damage
(Thousands of Dollars)

	Particulate Hatter Damage		
	Actual Baseline	Adjusted Baseline	Ozone Damage
San Jose			
Meeting Federal Standards			
Lower	587.0	0.0	3.5
Best	8646.7	0.0	11.2
Upper	16706.4	0.0	19.0
Meeting California Standards			
Lower	3526.1	2939.1	9.1
Best	51942.6	43295.9	29.3
Upper	100359.1	83652.7	49.6
Lowest Levels			
Lower	6300.8	5713.8	10.6
Best	92814.9	84168.3	34.2
Upper	179329.2	162622.7	57.8
Remainder of County			
Meeting Federal Standards			
Lower	6.6	0.0	5.2
Best	97.5	0.0	16.9
Upper	188.4	0.0	28.5
Meeting California Standards			
Lower	1598.3	1591.7	10.5
Best	23544.9	23447.4	33.8
Upper	45491.4	45303.0	57.2
Lowest Levels			
Lower	4357.2	4350.5	12.4
Best	64184.2	64086.7	40.2
Upper	124011.3	123822.9	68.0
Total County			
Meeting Federal Standards			
Lower	593.6	0.0	8.7
Best	8744.2	0.0	28.1
Upper	16894.8	0.0	47.5
Meeting California Standards			
Lower	5124.5	4530.9	19.5
Best	75487.5	66743.3	63.1
Upper	145850.5	128955.7	106.7
Lowest Levels			
Lower	10657.9	10064.3	23.0
Best	156999.1	148254.9	74.4
Upper	303340.5	286445.7	125.8

Table 7-2
Estimates of Ozone Levels for Each Scenario

Ozone Monitor	Baseline (Actual 1984 Levels)	Levels if Federal Standard Were Met	Levels if California Standard Were Met	Low Ozone Levels
ALUM ROCK				
Annual Average of All Hours	.0151	.0145	.0137	.0134
GILROY				
Annual Average of All Hours	.0173	.0165	.0153	.0151
MT. VIEW				
Annual Average of All Hours	.0138	.0138	.0134	.0131
LOS GATOS				
Annual Average of All Hours	.0155	.0142	.0134	.0130
SAN JOSE				
Annual Average of All Hours	.0164	.0160	.0146	.0143

7.5 REFERENCES

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8.0 SUMMARY OF RESULTS

Air pollution damages to human health estimated in this analysis are measured in numbers of cases for particulate matter, ozone, and lead. Some of the health endpoints, and the welfare impacts to materials and visibility, are also expressed in dollar values. Table 8-1 illustrates the numbers of cases of health problems estimated ("best" estimates) for each pollutant under two scenarios. For particulate matter and ozone, the estimates are annual damages for 1985 or 1984 relative to what would occur if the Federal or California standards were met. The health benefits of meeting the Federal PM standard are zero, because the adjusted baseline PM levels do not exceed the Federal standard. The health benefits of meeting the Federal ozone standard are about one-fifth the benefits of meeting the California standard.

The estimates for lead require a somewhat different interpretation, because they are based on the elimination of all gasoline lead relative to 1985 levels. The results of the lead analysis suggest that significant effects are occurring as a result of current lead emissions from motor vehicles, even though the ambient air quality standards for lead are not exceeded. The most significant categories of lead effects in terms of number of cases are hypertension in men, neurological effects in men, and blood/FEP effects in children.

The sums of the annual dollar estimates of morbidity and welfare damages associated with particulate matter and ozone are presented in Table 8-2. The figures for particulate matter are based on the adjusted baseline. The results obtained for current levels of ozone, as compared to meeting the Federal standards, range from $550 thousand to $740 thousand, with a best estimate of $650 thousand. About 30% of the estimated damages relative to the Federal standard occur in the city of San Jose (which has 50% of the population). The estimates for attaining the California particulate matter and ozone standards range from $34 million to $185 million, with a best estimate of $107 million. About 60% of the effects are in San Jose.

Table 8-1

Illustrative Magnitudes of Best Estimates of Health Effects Due to Criteria Air Pollutants in Santa Clara County

	Cases/Year Reduced if Federal Standards Were Met	Cases/Year Reduced if California Standards Were Met
Particulate Matter[a]		
Premature Mortality	0	37
Emergency Room Visits	0	2,200
Restricted Activity Days	0	367,400
Ozone[b]		
Asthma Attacks	400	1,900
Respiratory Restricted Activity Days	13,100	74,400
Eye Irritation	24,700	99,000

	Total Cases Reduced if Gasoline Lead Were Eliminated
Lead[c]	
Hypertension	2,380
Neurological Effects	987
Renal Effects	96
Reproductive Effects	16
Blood/FEP Effects	1,220
Fetal	2
Anemia	4

[a] From Table 2-4
[b] From Table 3-4
[c] From Table 4-4

Table 8-2

Total Dollar Value Estimates for Morbidity and Welfare Effects Associated with Particulate Matter and Ozone

(Thousands of Dollars)

	Particulate Matter (Adjusted Baseline)	Ozone	Total
San Jose			
Meeting Federal Standards			
Lower	0.0	169.7	169.7
Best	0.0	201.2	201.2
Upper	0.0	232.7	232.7
Meeting California Standards			
Lower	17443.6	1074.0	18517.6
Best	63153.4	1286.0	64439.4
Upper	111542.3	1498.0	113040.3
Lowest Levels			
Lower	31826.7	9923.9	41750.5
Best	119869.4	13573.7	133443.1
Upper	212446.9	17223.5	229670.4
Remainder of County			
Meeting Federal Standards			
Lower	0.0	400.0	400.0
Best	0.0	454.4	454.4
Upper	0.0	512.3	512.3
Meeting California Standards			
Lower	14076.4	1705.9	15782.2
Best	40531.7	1991.3	42523.0
Upper	69246.6	2292.4	71539.0
Lowest Levels			
Lower	28377.5	11364.1	39741.5
Best	96924.5	15463.8	112388.3
Upper	169576.1	19658.9	189235.0
County Total			
Meeting Federal Standards			
Lower	0.0	569.7	569.7
Best	0.0	655.6	655.6
Upper	0.0	745.1	745.1
Meeting California Standards			
Lower	31520.0	2779.9	34299.9
Best	103685.1	3277.3	106962.4
Upper	180788.9	3790.4	184579.1
Lowest Levels			
Lower	60204.2	21287.9	81492.1
Best	216793.8	29037.5	245831.4
Upper	382023.1	36882.4	418905.5

On a county-wide per capita basis, the best annual damage estimates for particulate matter and ozone, relative to the Federal and California standards, break down as follows:

	Per Capita Damage	
	Federal Standards	**California Standards**
PM Morbidity	$0	$11
Ozone	$0.45	$ 2
PM Materials	$0	$48
Ozone Materials	$0.02	$0.05
PM Visibility	$0	$15

The estimates for particulate matter materials damage are the largest. The estimates for particulate matter morbidity and visibility are roughly comparable. The estimates for ozone materials damage are small compared to the other estimates.

The results of the carbon monoxide analysis are highly uncertain, but suggest that there are significant numbers of individuals at increased risk of some health effects due to current levels of carbon monoxide.

Appendix A
DOLLAR VALUE ESTIMATES FOR CHANGES IN RISKS OF MORTALITY

Placing dollar values on risks of premature death can spark a lot of emotional controversy. It is important to note that what is typically being discussed with respect to environmental hazards is small changes in probabilities of premature death for many individuals. The dollar values are based on estimates of the value to the individual of reducing his or her risk of death by a small amount, not on the total value of a human life. Still, for the population as a whole this does mean a certain number of expected deaths and a total dollar value, which is objectionable to some people. Dollar values for the estimated changes in risks of mortality due to exposures to particulate matter presented in Chapter 2 are therefore presented in this appendix rather than in the main text of the report. The dollar value estimates, although subject to considerable uncertainty as is discussed below, allow some comparison of the relative significance of the various categories of damages estimated for each of the pollutants using a common metric.

Available estimates of willingness to pay to prevent small changes in probabilities of death are based on situations where individuals are observed making tradeoffs between probabilities of death and some benefit, such as income. These kinds of estimates have been recently reviewed by Violette and Chestnut (1983) and Violette et al. (1986) for their applicability to environmental policy questions. Most of the empirical work that has been done has looked at wage premiums associated with different levels of on-the-job risks. The size of the average annual on-the-job mortality risk in most of these studies is about .0001 per person per year. The estimated values to each individual of reducing his or her annual mortality risk by .0001 are between $100 and $700 per year, based on the most credible results of these wage-risk studies. Some contingent market studies have also been conducted in which subjects are asked what they would be willing to pay to reduce, for example, their risks of fatal accidents at work or in traffic accidents. The most credible results from these studies fall into the range of $100 to $300 for an annual risk reduction of .0001 per person.

A value of $200 per person for a risk reduction of .0001 was selected for this analysis as a fairly conservative representation of the kinds of values seen in the literature. This number is used to illustrate the order of magnitude that could be expected for the mortality risks presented in Chapter 2. There is considerable uncertainty in this estimate due to the wide range of results obtained in the literature, and the differences between the nature and size of the mortality risk involved in exposures to pollutants versus on-the-job accidents or traffic accidents. It is more likely to be too low than too high, but this is not certain.

The dollar value estimates for the estimates of premature mortality cases reported in Table 2-3 are presented in Table A-1 The estimates of deaths associated with particulate matter imply average risks to each individual of less than .0001, although the elderly and those with chronic illnesses would be expected to be at greater than average risk. To illustrate the implications of using $200 per .0001 risk, consider the lowest (greater than zero) and highest numbers of deaths predicted in Table 2-3. The best estimate for meeting the Federal PM standard (based on the actual 1985 baseline) is that 3 deaths per year would be prevented in Santa Clara County. This implies an average per capita risk of .0000021 (3/1,398,500), and the estimated average dollar value per person of preventing this risk is about $4 ($200 * .0000021/.0001). The upper estimate for attaining low PM levels is that 104 deaths per year would be prevented. This implies an average annual per capita risk of .000074 and the estimated average value per person is $148 ($200 * .0000074/.0001). The best estimate for attaining the California PM standard implies a value of about $57 per person based on the actual baseline, and about $53 per person based on the adjusted baseline.

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Table A-1
Dollar Values for Predicted Reductions in
Mortality Associated with Particulate Matter
(Thousands of Dollars)

	Actual 1985 Baseline		
	Meeting Federal Standards	Meeting California Standards	Meeting Lowest PM Levels
San Jose			
Lower	0.0	0.0	0.0
Best	5901.7	46180.5	74077.4
Upper	9026.1	70629.0	113294.8
Remainder of County			
Lower	0.0	0.0	0.0
Best	66.6	52058.2	61775.9
Upper	101.8	34038.1	94480.8
Total County			
Lower	0.0	0.0	0.0
Best	5968.2	80218.6	135853.3
Upper	9127.9	122687.3	207775.7

	Adjusted 1985 Baseline		
	Meeting Federal Standards	Meeting California Standards	Meeting Lowest PM Levels
San Jose			
Lower	0.0	0.0	0.0
Best	0.0	40278.9	68175.7
Upper	0.0	61602.9	104268.7
Remainder of County			
Lower	0.0	0.0	0.0
Best	0.0	33971.5	61709.4
Upper	0.0	51956.4	94379.0
Total County			
Lower	0.0	0.0	0.0
Best	0.0	74250.4	129885.0
Upper	0.0	113559.4	198647.8